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to the
SCIENCE of MEDICINE AND SURGERY
by the
FACULTY
In Celebration of
THE TWENTY-FIFTH ANNIVERSARY
1882 —— 1907
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FOUNDING
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NEW YORK POST-GRADUATE MEDICAL SCHOOL AND HOSPITAL
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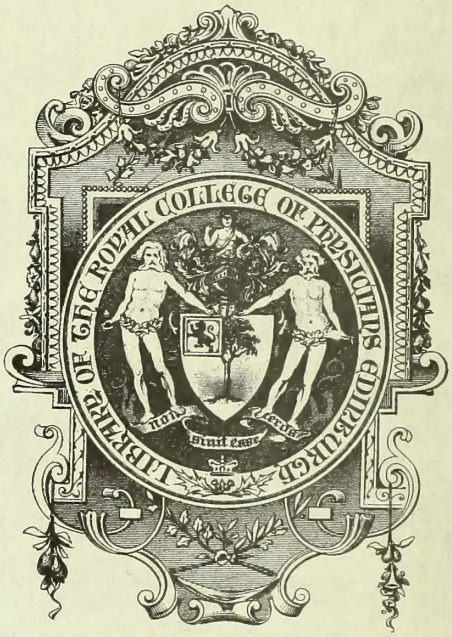
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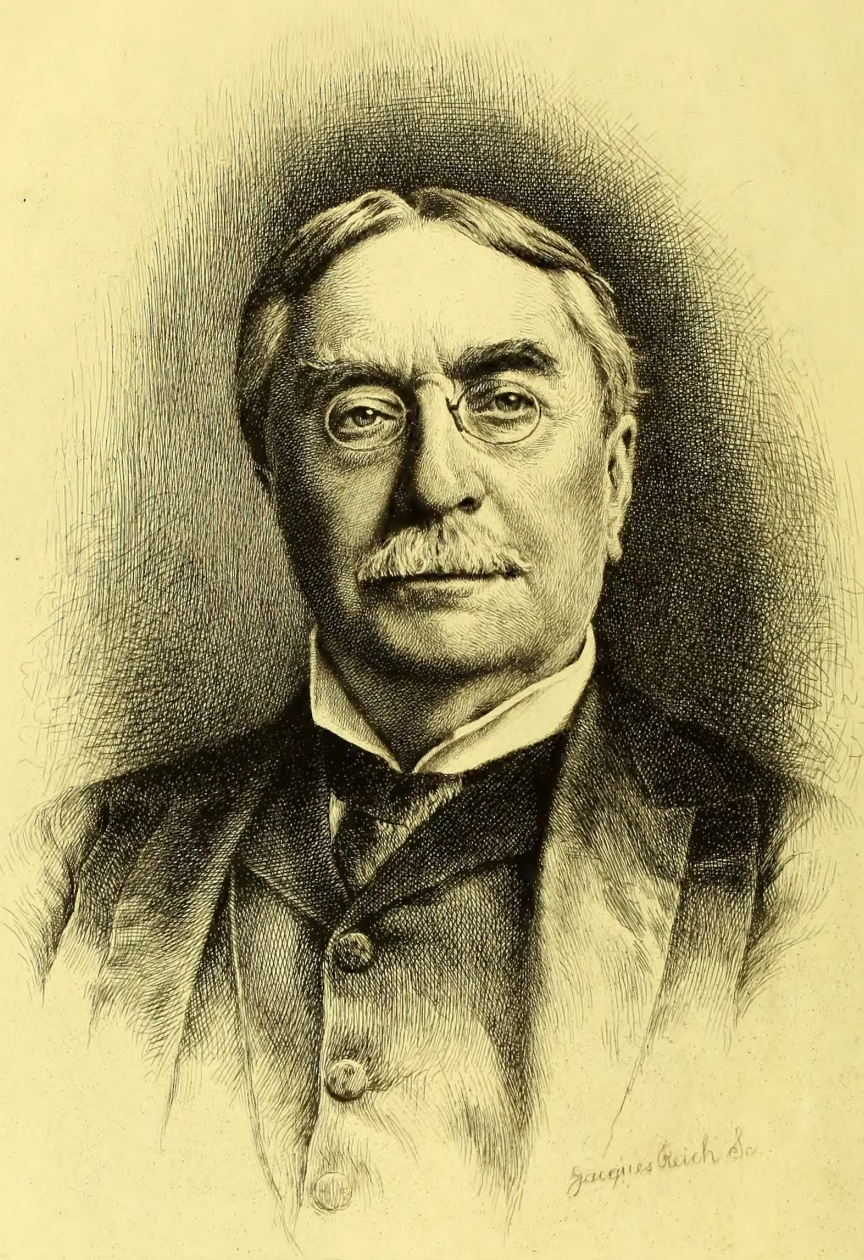

L.L.D. John Rogers

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to the

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TWENTY-FIFTH ANNIVERSARY

1882-1907

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New York Post-Graduate Medical School and Hospital

1908

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DANIEL BENNETT ST. JOHN ROOSA, A.M., M.D., LL.D.

Dr. Daniel Bennett St. John Roosa, President of the Corporation and Faculty of the New York Post-Graduate Medical School and Hospital, died suddenly, on March 8, at 8 o'clock, at his home, 20 East Thirtieth Street. Death was caused by heart disease. He was in his seventieth year.

Dr. Roosa was born in Bethel, N. Y., on April 4, 1838. He was of colonial descent on all sides of his family, and all his great-grandfathers were officers in the Continental line. His great-grandfather, Isaac, was a Captain in one of the New York regiments. Dr. Roosa was educated in the Bethel district school and at the academies of Monticello, N. Y., and Honesdale, Pa. He entered Yale in 1856, but was compelled to leave on account of poor health and studied under a private tutor until the following year, when he entered the medical department of the University of New York. He also studied chemistry under Dr. John W. Draper. He received the degree of M.D. in 1860, and in competitive examination won the place of assistant house surgeon at New York Hospital. The following April Dr. Roosa answered President Lincoln's call for volunteers and was appointed assistant surgeon to the Fifth New York Volunteers. Completing his three months' enlistment, Dr. Roosa returned to New York and finished his term of service in the New York Hospital as house surgeon. Leaving the hospital he went to Europe to study in the ophthalmic clinics in Berlin and Vienna. In June, 1863, he re-enlisted with the Twelfth Regiment of New York, returning at the end of his enlistment to take up private practice in New York. From 1863 to 1882 he was professor of diseases of the eye and ear in the University of the City of New York, now New York University, and from 1875 to 1880 he held a similar chair in the University of Vermont. He received an honorary degree of M.A. from Yale and an LL.D. from the University of Vermont.

He was the founder of the New York Post-Graduate Medical School and to date its only president. Had he lived until next month he would have celebrated his twenty-fifth year as head

of that institution, and the present volume was to be a testimonial to his work for education and medical science by his friends and pupils.

Dr. Roosa was president of the Medical Society of the State of New York in 1879, president of the International Otological Congress in 1876, and vice-president of the International Congress of Ophthalmology, at Edinburgh, in 1894. From 1893 to 1894 he was president of the New York Academy of Medicine. He was a member of the Union League and Century clubs and of the Holland Society, of which he was president from 1896 to 1897.

Dr. Roosa was the author of "The Old Hospital and Other Papers," "A Pocket Medical Lexicon," "The Determination of the Necessity of Wearing Glasses," "Defective Eye-sight," "A Treatise on Diseases of the Eye," "Otic Memoranda," "A Handbook of the Anatomy and Diseases of the Eye and Ear," and "A Treatise on the Diseases of the Ear, Nose and Pharynx," written with the coöperation of Dr. H. B. Douglass.

Besides these, Dr. Roosa wrote many articles for medical journals, and his editorial comments, which appeared monthly in the POST-GRADUATE, form a most interesting and illuminating contribution to current medical literature.

Dr. Roosa was particularly interested in state medical legislation, and was an active supporter of all those policies that tended to promote the welfare of the public and profession. At the time of the Code controversy, he was, with Agnew, Fordyce Barker and other leading physicians, most active and aggressive in promoting a more liberal and higher interpretation of what should be considered medical ethics and of the rules that should govern the conduct of professional men. His views are practically accepted now by the profession at large. The work he accomplished for the profession in antagonizing bad laws and promoting good ones has been widely acknowledged. A fitting tribute to this phase of his career was paid by Dr. Maurice J. Lewi, in the April number of the POST-GRADUATE. Dr. Lewi says:

> As indicative of the multitudinous topics which engaged Dr. Roosa's attention and which in turn were presented to the Legislature of the State of New York, either directly by him or through the Committee of which he was Chairman, the following may be quoted:

1. The transfer of the chronic insane from County asylums to State institutions.

2. " An Act to prevent blindness."

3. The regulation and the control of the embalming process.

4. Advancing the standard of academic requirements for all persons entering upon a career of medicine.

5. Safeguarding the interests of the New York State Medical practitioners as against practitioners resident in other States, desirous of being admitted to practice in New York State.

Dr. Roosa ceased his official connection with the State Society many years ago, but his interest in the welfare of the medical profession never lagged. He was ever ready to come to the rescue when danger threatened, and many a time, at a personal loss of income from his large practice, he went to Albany and to even greater distances to exert his voice and efforts for or against measures which had a bearing upon the well-being of medical practice.

Dr. Roosa was a Republican in national politics, and an active worker upon the committees of the Union League Club.

In 1904, physicians representing medical institutions from all the States east of the Mississippi and some from beyond, met at a dinner given in this city to honor Dr. Roosa and to commemorate the twenty-first anniversary of the inauguration in this country of post-graduate medical instruction. On that occasion a large loving-cup was presented to Dr. Roosa as the pioneer worker for post-graduate work.

At this dinner, Dr. Clarence T. Blake, of Boston, speaking of Dr. Roosa and his work, said:

> The great oak from the little acorn grows and spreads its branches wide, but it is necessary that the acorn should be sound at heart, that it should have a firm and wholesome fibre and that there should be within it the kinetic energy of growth—therefore, in behalf of a signal and original contribution to professional and public welfare, thoughtfully conceived, wisely planned and courageously sustained, let us pledge the warm heart, the firm-fibred intelligence and the well-directed energy of the president of the New York Post-Graduate School of Medicine.

Dr. William Osler, who presided as toast-master, introduced Dr. Roosa with the following tribute:

> You have worked hard, you have worked faithfully; you have been true to your ideals in medical education, and you have not neglected the duties of faithful citizenship.
>
> A rascally old astronomer physician of the seventeenth century wrote a delightful poem which has gone into literature as the " Wishing Song." It was the song which Benjamin Franklin says he sang a dozen times a

day as a boy, and as an older man he always regretted that so many of the wishes therein contained were denied to him. May I quote to you, sir, with slight variations, the concluding lines?

> " With a courage undaunted may *you* face your last day,
> And when you are dead may the better sort say,
> In the morning when sober, in the evening when mellow,
> He's gone and hain't left behind him his fellow;
> For he governed his passions with an absolute sway,
> Grew wiser and better as his strength wore away,
> Without gout or the stone, by a gentle decay."

The foregoing is a somewhat bare and inadequate account of the career and work of Dr. Roosa. It shows that his life was filled with many honorable achievements, but it does not tell us of that other silent part which a strong and inspiring personality plays in the development and help of forces about him. This is often the best that a man leaves behind: that he has influenced others for good and given opportunities and incentive to play a greater and more effective rôle in life. I do not feel that I am able to present in full and fitting terms a record of this character and its influence. I shall only try to pay here a somewhat informal and personal tribute to his memory, for I knew Dr. Roosa as a friend and physician better, perhaps, than in his other phases. There were, perhaps, particularly warm and kindly feelings between Dr. Roosa and myself, because he was a man of war, and I a man of peace. I admired and also wondered at his broad ambitions, and indomitable confidence which produced results at so much cost to his own heart's blood and personal ease. I felt a keen and genuine joy, which I knew he appreciated, to see him succeed in his great cause; against the odds of inexperience, stringent finances, and even personal antagonism.

Dr. Roosa was a most militant man, and a pioneer and idealist in his militancy. In earlier days he would have fought the savages and slain them; but he would have then established an institution and laws for them; and fought them again until they obeyed him for their own good. Resourceful, courageous, hopeful during the darker days, he both kept an even mind in adversity, and was wisely serene in success. To me Dr. Roosa always appeared a man of singularly open and receptive mind, despite the tenacity with which he held to his

opinions, and his course of conduct. For he was *tenax propositi*. Yet, knowing him for a quarter of a century, I always found that whenever a wisely thought out scheme or view was presented, it got his sympathetic attention, and candid judgment.

There were not many men of the breadth, and force, and training, or of the social and scientific attainments of Dr. Roosa. Therefore, he had nearly all the prizes that come in a medical career; and they came to him because they were due him.

Dr. Roosa was a pioneer amongst the otologists, as he was also among the ophthalmologists. The measure of his skill and scientific attainments I shall not attempt to estimate now. By associating with them larger activities than are called for by special clinical work, he set a fine and inspiring example to all.

As a practitioner and a consultant, he was wise, sensible, and straightforward in his opinions. He had his views, yet few prejudices. His attitude and treatment of his patients were those of a kindly and courtly gentleman. He was no warrior to those who were ill. In medical statecraft he brought about, as has been shown above, much legislation of permanent value to the profession and the public.

It is as a teacher and a promoter of a distinct advance in medical education that he will best be known, and as such he has achieved a permanent place in the history of medicine here; and of those endeavors to lift medicine to a higher plane of usefulness and esteem.

Dr. Roosa believed in doctors; he believed in the capacity of his own profession to do things, and to manage affairs. This was a part of his gospel; he preached it often; he told the doctor to believe in himself. Because of his insistance upon this gospel he became a personal uplifting force in the profession of this city and state, and, I may add, in the country at large.

I salute the memory of Dr. Roosa. We shall remember him for his militant spirit; his robust ambitions; his practical ideals. We shall remember him for his wit and humor; and his ready eloquence in debate; and his love of letters. Most of all, for the honesty of his work; his faith in God; in his fellow physicians; and in the institution for which he worked so bravely and successfully.

The sadness comes in knowing that we have lost the com-

panionship and helpfulness of a strong, high character; and a real and a very human personality. The memory of his character brings to mind lines which fit some, at least, of his activities and words:

> " To set the cause above renown,
> To love the game beyond the prize,
> To honor, while you strike him down,
> The foe that comes with fearless eyes
> To count the life of battle good,
> And dearer yet the brotherhood
> That binds the brave of all the earth."
>
> CHARLES L. DANA.

REPORT OF A CASE OF MASTOIDITIS, COMPLICATED BY PURULENT MENINGITIS, ENCEPHALITIS, PHLEBITIS OF SIGMOID SINUS, JUGULAR BULB AND INTERNAL JUGULAR VEIN, OPERATION, RECOVERY.

BY JAMES F. McKERNON, M.D.

Professor of Diseases of the Ear, New York Post-Graduate Medical School and Hospital.

The following case is reported because it presents several interesting features, especially the meningeal and brain complication which the otologist not infrequently meets with, and seldom with recovery.

The patient, a male aged 20 years, was seen in consultation with Dr. H. D. Chapin, on March 31, 1905. The following history was obtained from his mother. He had always been well except for the usual diseases of childhood, and a severe attack of articular rheumatism, when he was 12 years of age. Two weeks before he had an attack of follicular tonsillitis, lasting four days, and one week later he complained of severe pain in his ear, lasting four days, during which spontaneous rupture of the drum-membrane occurred. This was followed by partial relief for five days, when pain became severe again, and he was taken to an aurist who incised the drum-membrane, when he was again relieved of pain for several hours. This recurred again during the early hours of the following morning, and was quickly followed by severe pain, referred to the right side and top of the head, vomiting, dizziness, restlessness and groaning, and intense thirst.

Physical examination showed a well-developed adult, of 20 years, who was exceedingly restless, tossing from side to side of the bed, his eyes tightly closed, both hands clenched and emitting groan after groan with at times a high-pitched, inarticulate sound. Upon opening the eyes he would cry out with pain; the pupils were irregular with but little reaction, the left being smaller than the right; tongue dry and glazed, as were also the lips. The pulse was 100 per minute, full and bounding. Rectal temperature was 105.4 F.; respiration 24. He would lie quietly as though in a stupor, when suddenly he would arouse, ask for water, to again become exceedingly restless.

Aural Examination.—In the right auditory canal was found a bloody serous discharge, which, upon removal, disclosed a bulging drum-membrane with a small opening in it just above the region corresponding to the Eustachian entrance to the middle ear. The postero-superior canal wall was in a state of prolapse, hiding from view the postero-superior quadrant of the drum-membrane. Pressure over the right mastoid caused the patient to cry out with great pain, but upon further investigation it was found that pressure upon the other mastoid, or any part of the skull brought forth a like response. The left ear was negative. A diagnosis of mastoiditis with meningitis was made and an immediate operation advised as the only probable means of saving his life. It was explained to his family that in his present condition, recovery following operation was extremely doubtful. They, however, requested that every effort be made to save his life, and within an hour he was removed to the Post-Graduate Hospital and prepared for operation. A smear from the right auditory canal was taken to the laboratory for examination, and a report quickly given that the infection was that of the diplococcus intracellularis meningitidis. A differential blood count also was made, which showed a polynuclear percentage of 94, with a leucocyte count of 11,600.

Operation.—Chloroform was given, and the usual mastoid incision made. When the flaps were retracted, the external cortex was found to be very dark, almost black in color. Upon opening the mastoid it was found infiltrated throughout with a thin, colored serum, with here and there a cell containing thin milky-looking pus. The large medullary spaces posterior to the sigmoid groove contained the same characteristic pus and serum as that found through the mastoid proper. The bone comprising the zygomatic ridge was extremely cellular and found infiltrated, and was removed in its entirety from a point well forward anteriorly to a considerable distance further back than where the sinus passes transversely toward the torcular, this removal exposing necessarily the entire floor of the middle fossa in this region. The dura thus exposed was bulging and showed an intense redness over its whole area. It was found impossible to remove all the diseased bone without making a transverse incision posteriorly, and when divested of its bony covering the entire descending limb of the sigmoid sinus as well

as the dura over a portion of the cerebellum was exposed, and here it was free of congestion as compared to the dura covering the exposed area of the middle fossa above. After completing the operation, the exposed dura of the middle fossa corresponding to the roof of the mastoid antrum was incised horizontally over the bulging area, with the result that several drachms of turbid fluid were evacuated. The dura flaps were retracted and the brain tissue beneath inspected, with negative result. The opening in the dura was not closed; the entire cavity was packed with iodoform gauze, and the patient returned to bed in good condition. The duration of the operation was less than an hour. The pus taken from the mastoid, as well as the fluid evacuated through the opening made in the dura, was examined and found to contain quantities of the same characteristic infection shown from the smear examination, namely, *diplococcus intracellularis meningitidis*. As the temperature, respiration and pulse is here appended, I will not specify in detail as to their variations, as a study of the chart will show them. For several days following the operation the patient was never conscious, except once for a few minutes. This was on the fourth day early in the morning, and lasted only about five minutes, during all of which time he exhibited many, if not all the symptoms of a typical meningitis. The external dressing on the mastoid had to be changed twice a day, owing to their saturation, which undoubtedly came from the opening made in the dura. The wound itself was dressed on the third day, showing but little if any repair. Ice caps were kept on almost continuously up to this time. Several lumbar punctures were made, the amount withdrawn showing on the chart. All of the fluid obtained by lumbar puncture was turbid and showed quantities of the same infection as that before examined. The pupils were unequal throughout the entire seven days. The eye-grounds were examined three times during this period with negative results. On the eighth day following the operation, the patient regained consciousness; the ice caps were removed and kept off two days, to be reapplied upon his complaining of severe headache in the region of the wound and vertex. The wound was dressed every other day, and both the external dressing and packing were found saturated with fluid, but less in quantity at each subsequent dressing.

On the ninth day following the operation, the patient complained of feeling cold, and within a few hours exhibited a rise of temperature to 104.8° F. Believing we had a sinus phlebitis to deal with, I asked permission to explore this blood channel, but was requested by the family to wait. During the next four days there were no rapid excursions of temperature, the variations not exceeding two degrees. There were, however, several evidences of a chill, some vomiting every day, and the patient began to look septic. The wound upon dressing at this time showed but little repair, and where before the dressings had been saturated, they were now almost dry. That portion of the sinus divested of its bony covering did not exhibit its usual lustre, was somewhat grayish in color, and looked flatter than the average sinus when exposed. The blood examination at this time showed, upon differential count, a polynuclear percentage of 90.8, with white cells numbering 7,600. The following day the patient's condition was about the same as the four previous days, except that he complained of pain in the neck of the affected side. Physical examination here disclosed tenderness along the anterior border of the sterno-mastoid muscle, and some enlarged glands could be felt. Again operation was advised and this time consented to.

Second Operation.—Chloroform was administered and the sinus exposed one and three-quarter inches posterior to the knee above, and down to the bulb below. It was covered throughout its entire exposure with a grayish exudate, except the last half inch exposed. A free incision was made with a scalpel in its anterior wall at about the centre of exposure, and a few drops of pus exuded. There was no bleeding. The operative field was covered with gauze and the neck prepared for the removal of the vein. The internal jugular was exposed, ligated and resected from the clavicle below, to its exit from the skull above, as well as a portion of its tributaries, which were found involved. The lower two inches of the vein contained fluid blood, while all above this was filled with clot. The walls of the vein below at the clavicle were not thickened, macroscopically speaking, but above, just external to the foramen, the walls were found very much thickened. Several enlarged glands were encountered during the course of the dissection, and removed. The neck wound was then protected with moist

NAME.

DISEASE

1865 April

DATES OF OBSERVATIONS.

March 31 | April 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30

Cent. Fahr.

Pulse per minute.

Respirations per minute.

Urine Qnt. Spec. Grav.

(M'Kernon)

warm towels, and the contents of the sinus above evacuated, where plenty of pus was found, and a large amount of broken-down and disintegrated clot. Working back toward the torcular, a firm clot was found completely filling the lumen of the vessel, and upon its removal a free hemorrhage was established from this end. The disintegration below, above the bulb, was very marked, the broken-down clot with pus being about the consistency of thick molasses, and of a reddish brown color. A sufficient portion of the bony wall of the bulb was removed posterior, and below the facial canal, so as to permit the passage of a curette into the bulb, and after but little manipulation, free hemorrhage was established in this locality, the origin of the blood being, of course, from one or both petrosal sinuses. The hemorrhage was apparently as free as we would expect to find it, were the vein below intact and performing its usual function (a point which should not be forgotten), as many operators tell us that in phlebitis of the sinus proper, where a free hemorrhage is established here, it is not necessary to prolong the operation, as the blood which is present comes from an unobstructed vein below. This demonstration, as well as many others that have been made, show us, I believe, conclusively that the source of such bleeding is from the petrosals and not the vein below, as we may have a vein obstructed and still obtain abundant blood from this region. There was considerable sloughing of the posterior sinus wall, extending as far back as the cerebellum, and this was removed by the scissors. The wound above was dressed in the usual way; the neck wound was flushed with a hot saline solution; a cigarette drain was introduced, the opening closed with a continuous silk suture, dressed with gauze soaked in a warm saline solution, and the patient returned to bed in a rather poor condition. For the following two days his condition remained so, although he was conscious and took a fair amount of nourishment. A study of the chart will show that, three days after this operation, there was a rise of temperature to 102.8° F. Both neck and cranial wound were now inspected, the former being found in good condition with primary union from end to end. The cranial wound, however, was not found in as good condition; as over the cerebellum, where the sloughing edges of the dura had been cut away, there was a protrusion about the size of

an English walnut into the mastoid cavity of the cerebellar tissue divested of its covering. This mass was very soft and pulpy, and was removed with the scissors down to its base, and a drain, composed of a wick of gauze, was introduced into the cerebellar tissue for about three-quarters of an inch. The wound was dressed each day only to find mass after mass of disintegrated cerebellar tissue protruding into the mastoid wound. Upon three different occasions these masses were incised freely with the scissors deep into the cerebellum, but in a few hours their place was taken by more tissue of a like character. A further study of the chart will show that, for five days after the development of this encephalitis, there were wide and rapid variations of temperature, but no evidence of a chill was observed. On the seventh day following the second operation, the wound looked quite as unpromising as on the previous days, and after incising and removing more cerebellar tissue, a cotton-tipped probe was dipped in a solution of silver nitrate, of the strength of 480 grains to the ounce, and the entire cerebellar cavity that was exposed was freely mopped with this application and a large amount of boric acid placed in the cavity, and on this a very loose dressing of gauze was applied. From this time on there was no protrusion beyond the surface of cerebellar tissue; there was, however, an abundant slough as the result of this application. The cavity in the cerebellum began to fill slowly, so that at the end of ten days it was even with the surface, and each time the wound was dressed, an application of 360 grains to the ounce of silver nitrate was made, so that at the end of two and a half weeks the surface presented a rather firm cicatrix; and later, it protruded somewhat anteriorly, but always maintained its firm, scar-like appearance on the surface, and finally as the wound filled in, became covered with skin, and to all appearances, on palpitation and otherwise, became perfectly firm. The neck-wound healed without further complications, and one year after operation the hearing on the affected side was normal. The mastoid wound presents the usual post-operative appearance, with seemingly no more fulness than the average. The suture line in the neck, however, presented somewhat of a keloid appearance at this time. I think that there can be little doubt that this was a case of purulent meningitis, plus the other complications

that were present, as cultures made from the fluid evacuated through the dural opening, as well as those made from the fluid obtained by lumbar puncture, showed a true growth of the *diplococcus intracellularis meningitidis*.

This brings up the question of early operation in cases of purulent meningitis. It would seem rational to operate early, drain these infected areas, and treat them on general surgical principles rather than to stand idly by and see the vast majority terminate fatally. I believe that in the future this should be done, for even though but one case in several recovers, this in itself is a distinct gain, for now they are nearly all fatal. With improved surgical technique, may we not hope for more favorable results in the treatment of this condition?

62 West 52d Street.

HEPATO-INTESTINAL-TOXEMIA.

BY EDWARD QUINTARD, M.D.
Professor of Medicine, New York Post-Graduate Medical School and Hospital.

During the past few years a number of cases have come under our observation which presented such a definite symptom-complex that, for clinical purposes, it seemed practical to view and treat them as a class. Previous to this we had observed, from time to time, individuals suffering from symptoms presently to be described, but we had not observed a sufficient number, nor were we prepared to make an attempt at classification.

The classification, however, has been of such practical advantage to myself and certain of my colleagues who have seen fit to adopt it, that it seemed expedient to give a preliminary survey of such cases which, according to our idea, should be placed under the category.

At the outset we would state that the term hepato-intestinal-toxemia is a purely arbitrary one; a *generic* term, to be used for convenience until further and improved investigations and laboratory methods shall give us fuller and better knowledge and permit us to speak in *specific* terms.

We, ourselves, have not been able to find a term in medical literature comprehending the symptom-complex in question. Under such terms as "biliousness," "autointoxication," "intestinal toxemia," etc., we find a certain number of cases resembling those we are about to describe; but so far as we can judge, whether from omissions in the taking of histories, or from the lack of subsequent observations, the clinical pictures of such cases are vague and unsatisfactory. There seems to have been no attempt to classify them under a *generic term* or to depict a symptom-complex which would permit them to be so placed. In the present instance, we shall make no attempt to discuss the theory or origin of hepato-intestinal-toxemia. The purpose of this preliminary paper is a purely clinical one. We merely wish to present the symptom-complex of such cases. Without entering into the etiology of these cases, we may state that sufficient clinical facts are in our possession to warrant the conclusion that the symptom-complex in question is due to a toxemia in conse-

quence of faulty metabolism originating in the small intestine and its glandular appendages—the liver and pancreas.

We have seen cases at all ages and in both sexes, but the large proportion were between thirty and fifty years of age. Men seem to suffer more frequently than women. Cases are both acute and chronic, but the majority were ailing from the chronic course of the complaint; the condition extending over a period of many weeks or months, and not infrequently covering a period of years.

For the most part, and briefly stated, their condition, when they present themselves to the clinician, is as follows:

Their general appearance may be very fair, and to all intent and purpose, little seemingly ails such patients, their symptoms being subjective rather than objective. Such is the case, however, only with those who suffer from the milder degree of the condition, or with one in whom it has lasted but for a short period. These patients frequently present the appearance such as is seen in a mild grade of anemia, but, in addition, they are sallow. The conjunctivæ are slightly icteroidal. The term, jaundiced, does not apply to such patients, whereas sallow and icteroidal does. The tongue is coated yellowish white, or yellowish gray. This coating may be thin or thick; extend over the entire tongue, or be limited to the back of that organ. Often the tongue is dry. These patients may be fairly well-nourished, but in the chronic cases, where the toxemia has seriously affected the general metabolism and nervous system, marked emaciation may exist.

They not infrequently present a rather melancholic, listless, or depressed attitude, although, at times, the reverse of this is so in such cases as are complicated by neurasthenia. On questioning these patients, it will be found that they have certain characteristic symptoms; barring some of the very severe or, at times, unusual ones, these symptoms begin in a characteristic way and run a definite course. In other words, we may say that the *quality* of these patients' history is the same, although the *force* or *quantity* of any particular symptom may vary, and be more or less in any particular individual, the variation depending, to a great extent, on the patient's peculiar susceptibility to the toxemia and especially on his nervous condition at the time; for when hepato-intestinal-toxemia

has existed for any length of time, the nervous system suffers, and to the symptoms caused by the chronic poison must be added those of the neurasthenia, or will. This, at times, complicates the symptom-complex, and only experience and a thorough knowledge of the history of the case and of the conditions in question will permit one to say whether the complex of symptoms so frequently met with in these cases is due to the hepato-intestinal-toxemia, or to the neurasthenia; at times, indeed, the so-called " vicious circle " stands boldly in evidence, and it is quite impossible to decide this question.

The patients state that their condition is one of constant ill-health, with certain periods when they feel particularly bad; or else, they feel in good health for a few weeks, or possibly months, and then come attacks which render them wretched for a longer or shorter period. In other words, their condition is a remittent or an intermittent one. In the intermittent cases the onset of the attack may be preceded by a twenty-four hour period of what may be termed hypereuphoria, if such a term may be used to express an especially marked feeling of " *bien etre* "—a period when the patient feels more than ordinarily buoyant and energetic, with an increase in appetite and a desire to be up and doing. Undoubtedly, in such cases this is the first effect of the toxemia, a stimulation seen before the secondary symptoms begin. This period of hypereuphoria may be altogether absent. The following symptoms then become manifest, at times one and again another being in primary evidence:

An indescribable discomfort in the right, upper, abdominal quadrant. It is not a pain, nor is there any particular spot which in this region is very sensitive to pressure; yet the patients frequently complain that they feel as if they wished to " get away from something " in this region, and at times they will exert deep manual pressure over this area in order to see whether, by so doing, this peculiar discomfort can by this means be relieved. It would seem that, possibly, a mild duodenitis exists in such cases. Be this as it may, this symptom is a fairly constant one and may precede all other symptoms by twenty-four hours, or even somewhat longer. At times, however, this symptom is altogether lacking.

Circulatory and respiratory disturbances may be absent, slight or severe. Very frequently dizzy sensations are present,

and also feelings of "light-headedness." This dizziness, or "light-headedness" may be limited to such times when the patient makes a sudden effort to rise from a reclining or sitting posture, or when he raises the head suddenly from the inclined position. At times, however, these feelings of dizziness and vertigo are much more severe. They may, indeed, for the time being, prevent the patient from raising his head at all, or they may temporarily interfere with his gait. He may bump into doors and walls whilst walking, and this, combined with his other ill feelings, may alarm him and his friends. These disturbances are apt to be repeated in the severer cases, and in the remittent type may occur off and on, for weeks and months, or until such time as the toxemic condition has been cured.

Palpitation may come on at any time, especially from exertion, or after meals; but, often enough, the palpitation seems to be caused by the toxemia itself, and its time and mode of onset is variable.

Sudden dyspnea may occur in these cases, accompanied by feelings of impending dissolution. At times such attacks are severe and alarming, and their onset so sudden and unexpected, that it is difficult to persuade the patient that neither his heart nor his respiratory tract are diseased. In fact, the medical attendant himself, has he not seen such cases, may, until he has convinced himself by most thorough and repeated investigations, be led to believe that some obscure cardiac lesion exists.

In these attacks of dyspnea the patient feels that he cannot take a deep breath; his heart may palpitate; arythmia may occur, and to these sensations is added one of impending death. The patient may wake up with such an attack, or he may be walking in the street when seized with such a paroxysm. The first sensation may be that of a warm wave or flush passing suddenly over his entire body. Immediately upon this, he will be seized with the attack of dyspnea, palpitation, oppression in the chest, and feeling of imminent death. In some, dyspneic attacks may occur but once, or again, they may occur quite frequently during the course of the disease, and last for from a few minutes to an hour or longer. At times breathlessness on exertion is another marked symptom of both the acute and chronic form of the condition. Oddly enough, in cases in which it is manifest, it becomes especially marked after a dose of a mercurial, particularly after calomel. It would seem

that in such cases the sudden unloading of the poison from the liver and its reabsorption from the intestinal tract into the circulation, increase, for the time being, the toxemia, and so affect the neuromuscular system that any increased demand in the way of effort or exertion disturbs the respiratory and circulatory equilibrium.

Such attacks are more apt to occur in neurasthenic patients, but of their toxic character, in the mind of the writer, there exists no doubt. In the near future we hope to say something further as regards the toxins occurring in such patients.

Nervous Symptoms.—These are varied, and mild or severe according to the degree of toxemia and the susceptibility of the nervous organism of the patient. Headache of the migrainous type occurs, yet it is by no means a common symptom. The feelings of congestion: fullness and lightness in the head, especially a dull, heavy ache in the occipital region, are common symptoms, not only before and during the attack, but, in the remittent type, a rather constant symptom.

Aside from the temporary hypereuphoria mentioned as a prodroma, for the most part, the toxic conditions render these patients despondent and depressed. Indeed, when such a chronic toxemia is imposed upon an already neurasthenic or nervously-exhausted system, there is great difficulty at times to induce such patients or their friends to take a cheerful or hopeful view of the case; the marked depression and despondency causing them to believe that there is something ailing with their mind, or that they have an incurable malady.

At times, one of the most alarming symptoms, at least, so far as the patients are concerned, is thickness of speech and difficulty in the enunciation of words. This is especially apt to be the case when the patient speaks rapidly, and it may be that one especial letter or an especial word is more difficult for the patient to pronounce than another; as for instance, he may say " shlutter " for shutter, or " glass " for grass, and mistakes of a like nature; this occurring at the same time that the toxemia causes dulness and confusion of thought, which makes the condition seem one of very serious import. Such difficulty with speech, and confusion of thought, however, last only while there is an exacerbation in the toxic condition, and no ill-effects are manifest or left after this has been relieved.

Again, at times for months, these patients will feel giddy or uncertain when going down stairs. They feel as if they might pitch forward and fall. These sensations also are due to the toxemia and its effect on the cerebral circulation. They may also feel at times, when walking, as if the side-walk were suddenly coming up to meet them; this being a subjective symptom, pure and simple, and not an objective manifestation.

At times, however, it is really difficult, unless a most thorough examination is given these patients, to decide whether they may not be suffering from a far more serious lesion of the nervous system, or from a labyrinthian trouble. In a few of our cases, combined nervous and circulatory symptoms have been very severe; such as severe vertigo, followed by temporary loss of consciousness, and, in two instances, a mild, epileptiform seizure. This condition was but temporary, the patients coming immediately from the attack and having no further difficulty. These patients were exhaustively examined as to their condition, and it was only a knowledge of their previous history, and the notes from the specialists to whom these patients were sent for additional examination, that led us to view them as severe examples of chronic hepato-intestinal-toxemia.

Insomnia and bad dreams, especially the sensation of sudden falling, just as one goes to sleep, are quite common symptoms in these cases. It may here be well to state that, although we have these symptoms as a manifestation of a neurasthenic condition, and that in the instances where these symptoms were particularly manifest, the patients were, as a rule, neurasthenics, yet it could also not be denied that, when such symptoms occurred, there was every evidence of an increase in the toxemia.

Gastro-intestinal Symptoms.—So far as the stomach itself is concerned, in the vast majority of cases we find here no difficulty that can be made out, either by subjective or objective examination, with the exception of the loss of appetite which occurs during the exacerbations, or which may exist in the chronic type of the disease. Neither the physical nor the functional signs reveal anything abnormal. At times, to be sure, hyperchlorhydria may exist, and again, there may be hypochlorhydria. Occasionally there are evidences of gastritis, but we have never, for an instant, been led to believe that these cases were caused by any difficulty, primarily, in the stomach.

So far as the intestinal tract and liver are concerned, it is different. The at times marked constipation; the various fermentations; the evidence of intestinal putrefaction; the light, or almost clay-like, color of the stools (without jaundice and without bile in the urine); the poor absorption of fat; the discomfort in the right upper quadrant; all these symptoms point to the fact that the functions of the liver and intestinal tract are seriously disturbed. Indeed, the good results of treatment applied specifically to the liver and intestinal tract confirmed this view.

We have, then, in this intermittent type of the disease, periods of seemingly perfect health, interrupted by attacks having, as a rule, quite a characteristic onset, and lasting for from several days to as many weeks. Occasionally, however, the condition is one which, instead of ending at this time, runs a subacute course for a month or so; or at any one time, the intermittent type may change to the remittent type, when the clinical picture of the patients suffering from a constant toxemia, with marked exacerbations, becomes manifest.

The attacks in the intermittent type begin suddenly. The patient may have a period of hypereuphoria, when for a few hours to a day, he feels particularly buoyant and energetic; then he notices that his stools are much lighter in color; that he has a discomfort in the right upper quadrant of the abdomen; that his tongue becomes coated; his face sallow; that his conjunctivæ become icteroidal. At the onset he may suffer from polyuria, and the urine may be very light in color or be voided in much less quantity than normal. He feels decidedly depressed, and neuromuscularly weak; he may or may not feel drowsy; he may or may not have nausea; he very rarely has vomiting; his hands and feet become cold; he has chilly sensations, and it is frequently impossible for him to become warm; he has sensations of dizziness or of vertigo. He may have several or all of these symptoms. There may be numb sensations in the different parts of the body, especially, however, in the legs and fore-arms. These symptoms continue until the toxic condition is relieved. If the toxemia is not relieved, the cumulative effects of the poison appear, when the severer manifestations, such as marked dyspnea, severe vertigo, violent palpitation, feelings of dissolution, and numb-

ness of different parts of the body become evident. At times there is difficulty in enunciation of words. This is not a true aphasia, but the patient's tongue feels thick and he pronounces words wrongly, especially when speaking in a hurry. During such times, also, his memory is not keen. He cannot concentrate his thoughts, and at times, it is with great difficulty that he attends to his affairs. In other words, the patient seems to be drunk with the toxemia, and until he is relieved of this condition, any of these uncomfortable and at times distressing symptoms will continue.

In reference to the remittent type of case, it need only be said that the condition is, of course, a chronic one. It is just in these cases, however, where, at times, the differential diagnosis is exceedingly difficult and may be, for the moment, impossible. In fact, in cases suffering from this condition, more than in most diseases, experience counts for much. We have seen patients suffering from chronic hepato-intestinal toxemia, so sallow, emaciated and weak; with an almost cachectic appearance, and evidently so desparately ill that they presented the appearances observed in malign disease. Indeed, with such conditions, should there be hypochlorhydria, or concomitant myasthenia gastrica, with stagnation or possibly retention of food, it will take an expert to decide, at times, whether or no these patients are suffering from a malign trouble, and even then, decision must not infrequently be withheld for a while.

Temperature.—This varies considerably. More frequently than not, these cases have a normal temperature, with but a slight rise above or below the normal line. Cases are also met with, in which the temperature is higher—from 99.8 to 101° Fahr. and occasionally, even higher. These higher temperatures, at times, assume a typhoidal character and, in consequence, we have difficulty in distinguishing between certain of these cases and typhoid, or paratyphoid, or even obscure tubercular conditions. This is especially the case in instances in which the blood-count shows a leucopenia and a relative lymphocytosis. Such cases, however, are rare. They are mostly seen in cases of hepato-intestinal-toxemia in combination with severe neurasthenia, and it is in such cases, too, in which the afebrile type of the disease predominates.

Renal Symptoms.—The frequent manifestation of polyuria at the onset of the attack and the dark-colored urine voided as the

attack is passing off, are fairly characteristic. Traces of albumin, indican, occasionally urobilinuria or temporary glycosuria and disturbed ratios are frequent in these cases. On the other hand, the urine may present no change which ordinary laboratory methods can reveal. No doubt, however, exists in the mind of the writer that, with improved laboratory methods, we shall be able to demonstrate certain toxic products in this secretion. We shall discuss this more fully in a subsequent paper.

Treatment.—The first thing to decide is whether the patient should be put to bed or not. If the condition has existed for a long time, or if it is particularly severe; if the nervous symptoms are especially prevalent—particularly in the case of women; if the anemia is of a severe grade, or if such conditions as myasthenia gastrica, or concomitant gastrointestinal inflammation, exist, then these patients should be put to bed and kept there for from three weeks to two months. If possible, this rest-cure should be undertaken away from their own homes and immediate family, and no communication should be held between these patients and their friends until such time as the physician, in his judgment, deems reestablishment of such intercourse is permissible. Cases of hepato-intestina-toxemia complicated, as they frequently are, with neurasthenia and possible visceral disturbances, are difficult to treat, and unless, for the time being, their entire mode of life is under the strict surveillance of their medical adviser, such cases drift from bad to worse and, in the end, read disaster for doctor and patient alike.

It is well, at the onset, to inform both the patient and his family that it will take weeks and probably months for the patient to recover. In fact, it may take that length of time for him to get rid of the toxemia, and until this be done, success cannot be attained.

These patients should be thoroughly examined at the onset. This investigation, including repeated physical examinations, as well as those of the blood, urine, feces, gastric analysis, etc., can easily occupy a week. During such an examination the patients can be up and about, but if possible, it is far better to have them, during this period, either in bed or in a hospital or institution, where such examinations can be carried out in a thorough and systematic manner. Failure to cure these toxic conditions may frequently be traced to lack of thorough investigation at the onset.

Diet is an all important matter in cases of hepato-intestinal-toxemia, but we cannot emphasize too fully the fact that there can be no definite or dogmatic rules given to cover all cases. In other words, the results of the physical and clinical examinations obtained in each individual case must, in a great measure, be the guide. Certain general principles, however, can be laid down. The use of tobacco and stimulants should be positively prohibited. Coffee, as a rule, should be interdicted. Highly seasoned foods, condiments, salted, dried, smoked, or pickled foods of any kind should not be allowed. At times sweets can be given in moderation, as desserts in certain cases, but all such substances as candy, chocolate, cake, etc., should be forbidden. Sugar, taken in the morning on cereal, is a vicious habit, and if continued, it will cause gastral, intestinal and hepatic disturbances altogether unlooked for.

When in bed, massage, Swedish movements, hydrotherapy, are essential aids to the treatment. No patient suffering from hepato-intestinal-toxemia should be put to bed without having regular, daily massage; and yet, here it is has been our experience that there are certain exceptions, namely: in patients who are exceedingly nervous, it is, at times, wise to wait for a week to ten days before beginning massage; and, furthermore, in all cases it should be given mildly at the start. Disregard of this rule may cause very serious upsets at the beginning.

Everything for these patients should be according to rule, time, and definite system. Schedules for each patient should be made out and handed to the nurse. Under no circumstances should the patients be permitted to see either their charts or their schedules.

So far as drugs are concerned, our first object is to rid the system of the accumulated poison. This we do by flushing-out the liver and the intestines. Aside from especial treatment rendered necessary by definite gastrointestinal complications, it is the writer's treatment to give these patients, three times a day, or before each of the principal meals, 10 grains of glycerophosphate of soda in a large glass of hot water; to be sipped slowly. Care should be taken to select good preparations of glycerophosphate of soda, as many preparations socalled are very inferior. This especial part of the treatment can be kept up for weeks and months, with marked benefit. It is not only a good nerve-food, but it serves also to flush-out the gastro-

intestinal tract, and acts as a sedative as well. No result from this especial drug should be expected under four to six weeks.

It is also a habit of the writer to administer to such patients, once every other day for the first week, a mercurial in the form of the following pill:

℞

Ext. nucis vomicæ, gr. ⅛
Massæ hydrarg., gr. v.
Ext. colocyn. comp., gr. iij.
Ext. rhei, gr. vij.
M. et div. in caps. No. ii.
Sig: Take both capsules at night.

This pill is to be followed the next morning by a bottle of citrate of magnesia, or Rochelle salts, or phosphate of soda or its various preparations. An exceedingly good preparation of phosphate of soda is the socalled melachol, of which a tablespoonful may be given in a glassful of water, one-half hour before breakfast. Of course, on the morning when such salines are given, the morning glass of glycerophosphate of soda is omitted. The second week this pill is given twice a week, and the third week once.

It may be as well to remark here that blue mass is far more efficacious in these cases than calomel. The latter at times acts rather violently with these patients and leaves them depressed and with feelings of nausea and chilly sensations and muscular pains at times as long as forty-eight hours after giving the dose. Again, after calomel one is much more liable to have such symptoms as breathlessness on exertion than after the use of blue mass. Thus, treatment with blue mass and aperients may have to be repeated.

Especially beneficial in such cases are minute doses of calomel —1/40 to 1/20 of a grain—combined with a minute dose of salol, or carbonate of soda, or given alone. For several weeks at a time, it seems to act very well. Of the special gastro-intestinal difficulties which may arise as a complication; of these we cannot treat; it being altogether beyond the scope of the present paper.

It is well, also, if there is any colitis, or enterocolitis, or if constipation has existed for a long time, or if the toxemia is severe, especially if accompanied by intestinal flatulency, to give high colonic lavage. The solutions commonly used are normal saline solutions, or Carlsbad salts; of the latter a dram,

or two drams to the quart. The water should be fairly hot; otherwise, giving such lavages may cause severe abdominal cramps, and in these cases all unnecessary pain and excitement is to be avoided.

A word or two may be said about the anemia. After the first week or ten days, when, in other words, the liver and intestinal tract have begun to be flushed out, we may begin to give the blander preparations of iron. Of these the following are especially to be commended. Pyrophosphate of iron, in one grain doses in capsules or solution; peptomanganate of iron (Gude), or hemaboloids plain or arseniated. The iron tonic should not be given until the toxemia is at least partly under control. Later, when an iron-tonic treatment is necessary, the following combination will be found especially beneficial:

℞

Acidi arsenosi, gr. 1/30
Strych. phosphatis, gr. 1/40
Ferri pyrophosphatis, gr. j
M. et div. in caps. No. I.
Sig: Take one capsule three times a day after meals.

or

℞

Strych. glycerophosphatis, gr. 1/40
Ferri glycerophosphatis, gr. j
Mangan. glycerophosphatis, gr. ij
M. et div. in caps. No. I.
Sig: Take one capsule three times a day after meals.

For the nervous manifestations in these cases, especially at the onset, there is nothing better than the combinations of bromides. Five grains each of ammonium, potassium and sodium bromide, given in a little vichy or plain water two or three times daily, acts better in these cases of toxemia than any other sedative the writer knows. In cases complicated by neurasthenia, the preparations of valerian, especially valerianate of zinc, in 1 to 2 grain doses three times daily, seem to act well. Strychnin and its various combinations may be given with advantage where the patients are in need of such a neuromuscular stimulant and tonic, but if these patients are very nervous or the toxemia severe, strychnin should not be given at the start. The same may be said of arsenic and its various preparations.

NEURITIS AND ITS RELATION TO INTESTINAL PUTREFACTIVE PROCESSES.

BY GRAEME M. HAMMOND, M.D.
Professor of Diseases of the Mind and Nervous System, New York Post-Graduate Medical School and Hospital.

The more the putrefactive processes concerned in intestinal digestion are studied, the more we see its influence on diseases of the nervous system. We are beginning to believe that in many instances in which the etiology of some nervous affections has not been thoroughly understood, but which we believe to be toxic, can be directly traced to toxins which result from putrefaction of undigested material in the intestine.

It is the purpose of this article to show the relation of this process to neuritis, and to discuss the treatment best adapted for its relief. I do not purpose to review the work of the many investigators in this line, and the number is becoming greater every day, but to detail my own experience with a number of cases of neuritis, leaving the discussion of the relation of putrefactive toxins and the general nervous system to another occasion.

The following cases have been carefully studied and the evidence of putrefactive toxins as a causative factor has been clearly made out.

In the ten cases which I purpose to report, the neuritis was confined to a single extremity in six cases; to more than one extremity in three cases; and appeared as a severe multiple neuritis in one case.

Case I.—The patient, a man 45 years of age, had been engaged in hard physical and mental work for many years. He was moderate in the use of alcohol and tobacco. He never had any diseases at all, being a man of remarkable health and physical strength. He had been a very hearty eater for years. He had never had rheumatism nor any venereal disease. About a year ago he began to experience pain and weakness in the right hand. These symptoms gradually extended to the forearm, the arm and shoulder until the member was nearly paralyzed and exceedingly painful. There was tenderness of the nerve trunks, diminished reflexes, and a moderate degree of

tactile anesthesia. The body temperature was sub-normal, ranging from 96° in the morning to normal at night, reaching the normal point about nine o'clock in the evening; from that time the temperature gradually fell and reached its minimum of 96° F. at about six in the morning. Occasionally there were febrile exacerbations preceded by chills, followed by a mild fever, rarely going beyond 102°, and generally, but not always, followed by sweat.

Examination of the urine showed nothing abnormal except the continuous presence of indican and skatol. The examination of urine was made several times daily for over 30 consecutive days. In no instance did the urine fail to show a great amount of these toxins. Under treatment the patient gradually improved until at the present time the urine is free from putrefactive toxins, and has been for some three months. With the disappearance of the toxins the neuritis gradually improved and the patient is now recovered.

Case II.—A man 48 years of age, moderate in the use of alcohol and tobacco, and with no history of rheumatism, consulted me for pain and numbness in the left arm. The disease had begun by numbness in the fingers. Very soon pain began in the same region, and gradually extended up the arm, involving the entire arm, shoulder and pectoral region. At the time I saw him he had begun to have numbness in the right hand, the pain was very severe, continuous though variable. There was tenderness of the nerve trunks, slight tactile anesthesia and loss of muscular power. The reflexes seemed slightly exaggerated. There were no symptoms of rheumatism, nor of any other constitutional disease. Repeated examinations of the urine showed the presence of indican, and of other putrefactive products at every examination. Under treatment the patient has improved, and is now practically well.

Case III.—The patient, a man 54 years of age, whose life has been one of tremendous mental activity for many years, was referred to me by Dr. C. T. Adams. He had the most intense neuritis, limited to the right arm and shoulder. He had never taken alcohol to excess, and did not use tobacco at all. There were no evidences of rheumatism. The general symptoms of neuritis were well marked. The examination of the urine showed putrefactive products continuously. Under treat-

ment he recovered. He has had one or two mild relapses since, but in each case the same treatment has afforded him relief.

Case IV.—A man 44 years of age, three months ago began to experience numbness in the fingers of the right hand. Shortly after pain developed in both the radial and ulnar distributions. The pain was continuous, and gradually extended so as to involve the entire arm, the shoulder and the pectoral region. He had always been a strong, healthy man, had neither taken alcohol nor tobacco in excess; in fact had been very moderate in the use of both of these toxic agents. He had never suffered from rheumatism, nor did he in any way show any evidence of rheumatic disease. He had apparently no loss of muscular power. In his occupation he was obliged to lift heavy weights, and except for the pain occasioned by the use of the arm was as fully able to lift the weights as ever. An examination of the urine showed the presence of putrefactive toxins continuously. He has been under treatment only two weeks, and though slightly better has not by any means recovered.

Case V.—A man, aged 51, consulted me about a month ago for pain in his right leg, which he had had for six weeks. It began in the ankle and extended rapidly so that in a week's time the entire leg was affected. He also complained of some pain in the back in the lumbar region. He used tobacco freely, but was very moderate in the use of alcohol. He has never had rheumatism, has led an active life, and been free from any severe disease for a great many years. He had the general symptoms which distinguished neuritis. He had a great excess of putrefactive toxins, seen at every examination, and examinations were made frequently. Under treatment he rapidly recovered, and was entirely free from pain at the expiration of the fourth week.

Case VI.—A man, 52 years of age, a painter by occupation, who did not use either alcohol or tobacco, who said that he had never had lead colic or lead paralysis, and exhibited no signs of lead toxemia. He had never had rheumatism. Pain began in his left hip and gradually extended down the leg to the foot. All the symptoms of a very intense neuritis were present. The urine, examined frequently, showed a great quantity of putrefactive products. Under treatment he recovered in about eight weeks.

Case VII.—A woman, about 43 years of age, was taken ill with some kind of fever. She was not under my observation at the time, and I cannot give a more detailed description of her illness. Previous to the illness it was known that she had a sub-normal temperature. She had had a sub-normal temperature for many weeks. During this period of sub-normal temperature she was known to have putrefactive products in the urine continuously. Two or three weeks after her recovery from the fever she developed pain and numbness in both feet. This gradually extended up to the knees, and numbness and pain likewise appeared in both hands. She had never had rheumatism, nor had she at this time any symptoms of rheumatism. The urine was highly charged with putrefactive products when I saw her six weeks ago. She is still under treatment. The putrefactive products are disappearing from the urine, but the neuritis has not improved to an appreciable extent.

Case VIII.—A man, 44 years of age, who used tobacco and alcohol in moderation, and who had never had rheumatism or any serious illness in years, consulted me for pain, which had been present for two months, and which he stated began in both feet, extended up the calf of the legs to the knees, and the back of both thighs. He presented the general symptoms of neuritis in both legs. He complained of a little pain and slight numbness in the fingers of both hands. There was muscular weakness in both legs, but not in the hands. There was a great excess of putrefactive products in the urine. The patient is still under treatment. The toxins are disappearing from the urine and the neuritis is gradually subsiding.

Case IX.—A man, 58 years of age, had trouble for a year before I saw him with pain in the right leg, which was practically continuous. It had begun in the foot and had gradually extended until it involved the whole leg. Six months after the pain developed in the leg he began to experience pain in the left shoulder and arm. This had gradually increased in activity until at the time I saw him it was quite severe. There was no muscular weakness that could be appreciated. There was tenderness of the nerve trunks and tactile anesthesia. He had in former years been what he called a "free liver," indulging quite extensively in liquor and tobacco, but of recent years he had been extremely moderate in the use of both. He had never

had rheumatism. He appeared to be a man in excellent general health, well preserved. The urine was loaded with putrefactive products. The body temperature is generally subnormal, ranging from 97° in the morning to a little above normal at night. He is still under treatment, but as he shows little or no inclination to conform to dietary restrictions, there has been no appreciable improvement in his case.

Case X.—A woman, about 70 years of age, who never used alcohol at all, and who had never had rheumatism, and had enjoyed excellent health for years, gradually developed a neuritis, beginning in both feet, which gradually extended until it assumed the form of the multiple type. Her temperature was sub-normal, and she had occasional febrile attacks. Putrefactive products were found in great excess in the urine, and continued so in spite of all forms of treatment which were instigated, both dietary and medicinal. The disease assumed a very severe form until there was almost absolute paralysis of all the voluntary muscles, except those of the face and neck, with atrophy and general anesthesia together with the other symptoms of multiple neuritis. The treatment has been persisted in with indifferent result. She has been ill over a year, but for the past three months there has been gradual improvement. This was the most severe case that has come under my observation.

Analysis of these cases will show that of the ten, eight occurred in men, and two in women, and that all of them were in persons of middle life or beyond. This is to be accounted for by the fact that men commonly eat more in proportion to their weight than women do, and would also seem to show that putrefactive toxins are either not prone to cause neuritis unless they have been more or less continuously present for many years, or else that as the body grows old it possesses less resistance to toxic influences. In most of the cases there had been immoderate indulgence in food for years, and in all of them I was able to ascertain that the patient had suffered from intestinal indigestion for a long period before the neuritis showed itself.

From a study of my cases I am not inclined to the belief that indol alone is a very toxic agent. It is probable that there are some nervous organizations, which may become diseased by

the long-continued poisoning with this substance. I believe, however, that skatol and butyric acid are much more powerful toxins, and that it was to the presence of these toxins and possibly to others more than to indol, that the neuritis was due. The sub-normal temperature, which existed in some of the cases, and particularly in Case I, was a well-marked symptom, and was undoubtedly caused by chronic intoxication of many years' duration. I am inclined to believe that these toxins are well borne by the nervous system for a long time, and that it is only in continuous poisoning by these agents that disease of the nervous system ensues. In the treatment of this condition a physician encounters a difficulty of considerable magnitude in having to subject his patient to a strict dietary which most of the patients do not wish to carry out. Almost all of my patients were hearty eaters, who enjoyed their food, and who found it exceedingly disagreeable to follow my directions. In many instances there were relapses from indiscretions, which the patients' desire for rich food led them into.

It is commonly believed that proteid foods are the ones concerned in the formation of putrefactive toxins; that this is so in the main is true, but it is quite clear to me that all forms of food, when eaten in greater quantities than the digestive fluids can digest, are capable of forming putrefactive poisons, which are deleterious to the human organism. I have, therefore, insisted that all of my patients should eat small meals in which the proteid foods should form a very small proportion. To my mind, it is not so much a question of what the patient eats as it is how much he eats. I have found the best results follow when general food was given, but the quantity of food of any kind was limited to a very small amount. Most all people habitually eat, at least once a day, and frequently at all meals, not only much more food than they need, but much more than they can digest. In the latter case, putrefactive decomposition naturally follows. This may be obviated by giving only as much food as can be digested, and it is even better to give, for a time at least, even less than this amount. When this plan was followed every day for a number of days, the putrefactive products in the urine were invariably decreased, and in some cases entirely disappeared. In none of the cases did I confine the diet to sour milk in any of its forms, nor did I use

any other preparations containing lactic acid. Under this reduced diet, the heaviness and sleepiness, which were often an accompanying symptom, disappeared, the temperature became more normal, and the patient presented a far healthier general appearance.

Medicinally, I found the best remedies were sulphate of quinin and aspirin in combination. I generally gave from two to three grains of quinin with eight to ten grains of aspirin, three times a day. Sometimes I have given the quinin alone in doses of three to four grains, three times a day. It is possible that the quinin acts through its germicidal properties, and possibly the aspirin, being a salicylate, acts in the same way. At any rate, after employing a great many remedies my experience has led me to adopt these two as giving better results than anything else.

I am inclined to believe that alcohol in any form, but particularly with meals, retards the digestion in this class of cases. It is claimed that in the ordinary individual a moderate quantity of alcohol, taken with the food, accelerates digestion, but I firmly believe that this is not the case with those people who suffer from intestinal putrefactive indigestion. I have not observed that the moderate use of tobacco, in the form of smoking, has any effect in causing or continuing the development of intestinal toxins in the average individual, but the excessive use of tobacco is to be deprecated, of course, as injurious to the general health. In any case, I believe that the keynote to the successful treatment is the ingestion of small quantities of food at any given time, no matter whether the food be proteid or carbohydrate or hydrocarbon. Any one of these, if given in quantities more than the individual can digest, contributes its share toward the formation of some putrefactive product.

Thorough mastication of the food is imperative, and it is of great importance to see that the patient has a great deal of sleep, that the bowels are kept open and that he bathes daily. From the nature of the malady, physical exercise can seldom be indulged in, so that it is essential that the bodily health be kept at its best by the strict observance of all other hygienic procedures.

SOME MEDICO-LEGAL ASPECTS OF INEBRIETY.

BY G. ALFRED LAWRENCE, LL.B., PH.D., M.D.
Instructor in Diseases of the Mind and Nervous System, New York Post-Graduate Medical School and Hospital, etc.

The subject of inebriety or alcoholic addiction is one that is assuming greater and greater importance as the conditions of our advancement in civilization become more complex and thus produce ever-increasing demands upon the individual members of the social system. Even now, at the present session of the New York State Legislature, a bill has been introduced to create a commission to study the treatment of inebriates and persons addicted to the excessive use of narcotics and their relation to the commission of crime and, furthermore, as to the expediency of establishing a State Institution for the care of such persons. It is to be hoped that such a commission will be the means of establishing a suitable colony for inebriates under the supervision of skilled inebriatists (if we may use such a term to distinguish a physician especially trained in the care and treatment of inebriates), and along the broad scientific lines now so successfully carried out by the State at the Craig Colony for Epileptics at Sonyea, Livingston Co., New York, after sixteen years of existence. The unfortunate one who, by heredity or limited physical or mental endurance or from unusual demands upon him, physically or mentally, finds that by alcoholic stimulation the jaded and overtired body and mind can be whipped up to accomplish the task set before him, too soon in the great majority of cases, the ever-increasing amount of stimulation required renders him a helpless slave to the habit, broken in health, an economic burden to his family or the State and in many cases an offender of the law.

Of the many effects of inebriety, Mendel (1907) estimates that 15% of insanity is caused by alcoholism directly, to say nothing of a still greater number who, from the contraction of venereal and other diseases, the practice of various excesses, and from hereditary tendencies, finally become insane but in which alcoholism is but a secondary or contributing cause.

The New York State Lunacy Commission from 1888 to 1902, in their admissions to the various state hospitals for the insane, found

that 9% of all cases thus admitted were directly due to alcoholism. In France, owing to the very general use of absinthe in addition to other alcoholic liquors, 13.6% of all admissions to public insane asylums are due more or less exclusively to alcoholism and have thus become economic burdens upon the State. Nearly one half of this total number or 6.8% were absinthe drinkers. In the *French Official Journal*, for 1907, Mr. Mirman, director of the Department of Public Assistance and Hygiene, determined from statistics collected for the ten years, 1897 to 1907, that there was an increase of 57% in the number of insane in 36 departments of France during the past decade, thus showing a proportionate increase in inebriety. Again, in the dread disease epilepsy occurring in one of every three hundred persons, it is estimated that 14% of the cases are directly due to alcoholism, and we find it the direct or contributing cause in many other diseases, notably of the nervous system, the heart, kidneys, and liver. Furthermore, Sachs (1900) estimates that fully 50% of all crimes committed in Germany are due to alcoholic excesses, and, no doubt, the percentage is as high or higher in England and America, according to Hammond (1904). It is notorious that most prostitutes are alcoholic, which makes them still greater factors in the spread of disease and the perpetration of crime.

Many crimes which become of medico-legal importance are committed when the degree of intoxication is such that the inebriate is incapable of appreciating the nature and quality of his act, his reason being defective to that extent. Finally, in the causation of suicide, alcoholism plays an important rôle. During the year 1906, the total number of suicides in Prussia was 7298, of which 12.9% were directly due to alcoholism, and of these 11.6% were men and 1.3% women.

Inebriety, according to Crothers (1888), has been recognized as a disease ever since the second century of the Christian Era, when Vulpian, a Roman jurist, urged the necessity of treating inebriates as sick and diseased and with special surroundings and by special means. In 1747, Cadillac, of France, urged that the State provide special hospitals for dipsomaniacs. Since then Dr. Rush, of Philadelphia, in 1790; Dr. Cabanis, of Paris, in 1802; Professor Plateur, of Leipzig, in 1809; Salvator, of Moscow, in 1817; Esquirol, of France, in 1818; Crammer, of Berlin, in 1822, all in their time were active advocates of the treatment

of inebriety in suitable inebriate asylums. In 1830 the Connecticut Medical Society appointed a committee to report on the need of an asylum for the medical treatment of inebriates. In the same year, Dr. Woodward, of the Worcester Insane Asylum, Massachusetts, made a plea for special hospitals for inebriates and that inebriety be treated as a disease. In 1844 the English Lunacy Commission urged that inebriates be regarded as insane and sent to asylums for special treatment. In 1864 the first inebriate asylum was opened for patients at Binghampton, New York, but from lack of proper public support and political dissensions a few years later, it was changed into an insane asylum. Since then, however, numerous asylums for the treatment of inebriates have been opened in the United States, various countries of Europe, Australia and New Zealand and are doing important work in the solution of the proper care of this large class of cases.

For the purposes of this article, the term inebriety is used synonymously with drunkenness or habitual intoxication in which a sufficient quantity of alcoholic beverage—which may be very small or extremely large—has been imbibed to produce a psychosis. This, therefore, excludes all those who may drink even large quantities of liquor so long as the habit does not cause them to depart from their normal psychical state. In classifying the alcoholic psychosis we may have: (1) acute alcoholic psychosis; (2) subacute alcoholic psychosis; (3) chronic alcoholic psychosis, and (4) dipsomania.

(1) Taking up the first division more in detail, the acute alcoholic psychosis may be an exacerbation of a chronic alcoholic psychosis in which, at intervals, the chronic alcoholic may have an acute alcoholic condition and later, after a longer or shorter period, lapse into the chronic stage again. Furthermore, it may originate from the use of alcoholic stimulation in a debilitating somatic disease, especially where a considerable hereditary taint is present; also in epileptics, and after injuries to the cranium. The quantity imbibed, individual susceptibility, and environment are also important factors. There are several varieties of this acute alcoholic psychosis that should be noted: (*a*) apoplectic type, with unconsciousness; symptoms of general paralysis, collapse, and death; (*b*) convulsive type generating an epileptic seizure and maniacal state which may increase to raving (*mania acutis-*

sima ebriorum) with manifold frightful hallucinations, often leading to violent assaults or destruction of anything within reach of the maniac; (*c*) twilight states or alcoholic trance in which the inebriate appears calm, answers questions of an ordinary character without delay and correctly and to the unskilled observer appears normal. These cases are of especial medico-legal interest as they may, while in this state, do peculiar things, utter indecent expressions, commit indecent exposures, various crimes, and attempt suicide. On the other hand, they may remain in a state of stupor and become the victims of crime. Such an acute state of alcoholic psychosis may last but for a few minutes or continue for a day and usually passes off in a deep sleep.

(2) Of the subacute alcoholic psychosis, (*a*) delirium tremens, (*b*) alcoholic melancholia, and (*c*) alcoholic paranoia are the most important varieties. Delirium tremens may run the ordinary course with gastric disturbances, anxiety, trembling, insomnia, delusions with sense deceptions, frightful hallucinations, at times leading up to the highest degree of frenzy, epileptic spasms, mussitating deliria, and finally recovery in the course of a few days or weeks, or a chronic alcoholic state develops lasting for weeks with repeated relapses, or death may terminate the case. As variations we may have an abortive form of delirium tremens with hallucinations, but the patient may seem very intelligent and perform his accustomed labor, although it may be interrupted. Furthermore, we may have a chronic form in which the delirium drags along for many weeks with repeated relapses; also a febrile form with temperature as high as 103° F.; or an epileptic form in which epileptic seizures may occur frequently during the course of the disease. This latter form is not to be confused, however, with epileptics who contract the alcoholic habit and develop delirium tremens from the latter cause. The amnesia will determine this point. Finally, we may have the polyneuritic type or Korsakoff's disease of alcoholic origin, in which the symptom-complex of multiple neuritis, gastric disturbances, disorientation as to time and place, striking disturbances of memory, hallucinations, delusions, and illusions are the prominent symptoms. Sometimes a stuporous state supervenes instead of the delirium. In this type, the disease is usually protracted, often a year or longer, with final

recovery in most cases; in other cases a varying grade of alcoholic dementia develops, or a paranoid symptom-complex, and in some cases death terminates the case either from exhaustion or suicide (the latter in from 5 to 10% of the cases).

Alcoholic melancholia may arise after acute excesses on the basis of chronic alcoholism, in which the symptoms are a melancholic depressed state with self accusations, depressive delusions and hallucinations and symptoms of stupor which usually pass away in a few weeks.

Alcoholic hallucinatory paranoia (Wernicke's Acute Hallucinosis of the Drinker) is still another type of the subacute alcoholic psychosis, in which there may be ideas of detraction often combined with megalomania; vivid auditory hallucinations are especially prevalent, the contents of which may be severe injuries, words of abuse, and threats of severe punishment. These delusions develop especially in two directions: either in unwarranted jealousy (that wife is unfaithful, etc.) or religious delusions (called by God to do certain things). The course of this type is sometimes very stormy, with hallucinations of all the senses and violent emotions of apprehension, sometimes terminating suddenly in from one to four weeks; or it may continue for from six weeks to several months, while a small proportion of the cases go on to incurable chronic paranoia.

(3) Chronic alcoholic psychosis comprises those cases gathered from the first two groups that become chronic in character, and many others who, after years of constant drinking, develop mental weakness, lack of energy, limitation of judgment, weakened memory, disturbances of moral feelings and indifference to their own interests, calling, or friends. They become less scrupulous in their transactions and simply exist for the gratification of the passions, engage in immoral acts—begging, stealing, deceptions, forging checks, etc. These chronic cases may develop either the acute or subacute psychosis, previously described, in the course of their disease, and the final result may be: (*a*) improvement under suitable treatment and with proper environment with tendencies to relapse; (*b*) incurable mental disease, as alcoholic dementia or paranoia, and (*c*) death from organic changes which alcohol has provoked (heart, kidney, hepatic diseases, etc.), or by suicide.

(4) Dipsomania or periodical maniacal outbreaks, ac-

companied by immoderate thirst for alcoholic beverages, is usually found in inheritors, imbeciles with small psychical powers of resistance, especially when assuming new burdens; cases of periodic mania of alcoholic habits, maniacal phase of maniac depressive or circular insanity, of which the depressive state is not very prominent; periodic melancholia or hypochondria, in which stimulation is resorted to to relieve the depression; also periodic nervous diseases, on the basis of hysteria with heightened appetite for alcohol and total aversion to nourishment, of epileptic nature and may seem periodically like an epileptic dream-state. In the intervals there may be an absolute aversion to alcohol. Some of these cases develop delirium tremens, and others pass into a condition of chronic alcoholism.

From the above classification and symptomatology, it will be seen that there are many medico-legal complications that may result in the case of the inebriate and requiring the most careful search into the history, the physical condition and mental state of the individual prior, during and subsequent to the act; the quantity and quality of alcoholic stimulation imbibed, and accurate and complete knowledge of all the facts and circumstances leading up to, during and following the act, before a correct opinion can be given as to the responsibility of the individual. In addition to all this, we must take into consideration the attitude of the law regarding an alcoholic and his acts. Inebriety, in itself, is not considered a punishable offence against the State, but when the inebriate violates the rights of others or commits offences against the public, he will be held accountable for the same. The common law, according to Bouvier (1897), is not disposed to afford any great amount of relief, either in criminal or civil cases, from the immediate effects of alcohol, and it has not considered mere drunkenness *alone* as a sufficient reason for invalidating any act.

In England, drunkenness has never been admitted in extenuation for any offences committed under its immediate influence; and centuries ago (end of 16th Century), the famous Lord Coke said: "A drunkard who is *voluntarius daemon* hath no privilege thereby; whatever ill or hurt he doth, his drunkenness doth aggravate it." Occasionally, however, lawyers have shown a disposition to distinguish between the guilt of one who commits

an offence unconsciously, in consequence of drunkenness, and that of one who is actuated by malice aforethought and acts deliberately and coolly. Later, a case has held that drunkenness, by rendering the party more excitable under provocation, might be taken into consideration in determining the sufficiency of the provocation (7 C. and P. 817). In a later case it was declared that there might exist a state of drunkenness, which takes away the power of forming any specific intention (4 Cox. Cr. Cas. 55).

In the United States courts have gone still further, and it has been held that where murder was defined as wilful, deliberate malicious and premeditated killing, that the existence of those attributes is not compatible with drunkenness (13 Ala. N. S. 413). Also, that when the state of intoxication is so great as to render him unable to form a wilful, deliberate, and premeditated design to kill or of judging his acts and their legitimate consequences, then it reduces what would otherwise be murder in the *first* degree to murder in the *second* degree (29 Cal. 678). When one who intends to kill another, however, becomes voluntarily intoxicated for the purpose of carrying out the intention, the intoxication will have no effect upon the act (36 Pac. Rep. 770). Furthermore, if one person gets another drunk and persuades him to commit a crime, the *former* is responsible (91 Ga. 740). In another case (88 Ala. 100) it was held that intoxication does not excuse crime, but may show an *absence of malice*; but the burden of proof is on the defendant to show intoxication to such an extent as to render him incapable of malice.

In the matter of robbery, it has been held that if the act was committed while the person was in such a drunken state as not to know what he was doing, he will not be deemed to have taken the property with a felonious intent (92 Ky. 522). In a case where injury of the head had been followed by occasional paroxysms of insanity, in one of which the prisoner killed his wife, it appeared that he had just been drinking, and that intoxication had sometimes brought on the paroxysms, though they were not always preceded by drinking. The court ruled that if the mental disturbance was produced by intoxication, it was not a valid defense; and, accordingly, the prisoner was convicted and executed. The principle adopted here was that, if a person voluntarily deprived

himself of reason, he can claim no exemption from the ordinary consequences of crime (Ray, Med. Jur. 574).

Delirium tremens is classified as a form of insanity in which the party is not responsible for his acts, in Wharton and Stille's Medical Jurisprudence (1905). Mania a pótû (delirium tremens) is given as an excuse for crime, although drunkenness is not (100 N. C. 457). Such a decision, from a medical standpoint, is irreconcilable with the fact that mania a pótû is but a form of drunkenness. Another series of cases makes a distinction that, where dipsomania effects the intellect and not merely the will, it may be a defense (105 Cal. 486). Still another distinction is made that where a person, in regard to a particular act, though knowing right from wrong, has lost his power to discriminate, in consequence of mental disease, he will be exempt from his crime (115 N. C. 807). Dipsomania, in the present state of judicial opinion, would hardly be considered a valid defence in a capital case, though there have been decisions which have allowed it, holding the question whether there is such a disease and whether the act was committed under its influence to be questions of fact for the jury (40 Conn. 136). The law does, however, recognize two kinds of *inculpable* drunkenness: one produced by the "unskilfulness of the physician", and that produced by the "contrivance of enemies," (Russ, Cr. 8). To these may, perhaps, be added one in which the party drinks no more liquor than he does habitually and without becoming intoxicated, but which exerts an unusually potent effect upon the brain, in consequence of certain pathological conditions (5 Gray. 86). In the case of Arson, the distinction is made, and it has been held that inebriety is no defense where it appears that the act of setting on fire was wilfully done, it being of no consequence what the intention was (2 Edin. Sel. Cas. 80). In negotiable instruments, drunkenness is no defense as against an innocent purchaser for valin (91 Pa. 17). The inebriate's contracts are not void, but voidable only (8 Am. Rep. 246). When deprived of all consciousness, strong presumption of fraud is raised, and on this latter ground courts may interfere (1 Ves. 19).

Marriage is a contract, and if entered into by an inebriate lacking contractional capacity, is voidable but may be subsequently ratified upon returning to a condition of sobriety and thus make the contract binding. This statement also holds

good in the case of that form of inebriety desginated as dipsomania, where the contract is entered into during the period of alcoholic mania, but not so if entered into during the intervals.

Chronic inebriety, if shown to have affected the mind of the contracting party to the extent that he could not comprehend the nature of his act, also makes his marriage contract voidable. In the case of divorce, the laws of different states vary. In New York State, inebriety is not recognized as a ground for divorce, while in some states habitual drunkenness is so recognized (130 Ill. Rep. 230). In many states, legal separation may be obtained on the ground of habitual drunkenness, especially if it can be shown that the life, limb, body, or health of the other party is endangered. Adultery, rape, or other sexual acts committed by the inebriate, owing to delusions of a libidinous nature and uncontrollable impulses, are, as a rule, not valid grounds for divorce, but would be if it could be shown that there was no relation between the acts complained of and the delusions. In an action for breach of promise to marry, inebriety will constitute a valid defence if it can be shown that the contracting party did not know the nature and quality of his act, and such a contract will be voidable unless subsequently ratified after becoming sober. It has been held, however, that evidence that the plaintiff drank intoxicating liquors to excess, was not admissible as a defence (1 Abb. App. Dec. 282). Whether it will constitute a defence for the party afflicted, is a question of much difficulty unless it can be clearly shown, as stated above, that at the time of making such an executory contract the afflicted party did not know the nature and quality of his act, and if this latter is proved; it will not do more than make the contract voidable. If such contract, however, is obtained by fraud, by making the party purposely drunk, the transaction will be absolutely void, no contract having been entered into. If the defendant in such action did not know of the inebriety of the plaintiff at the time of making the promise, such a plea would be a complete defence, especially in the case of dipsomania or chronic inebriety. If inebriety results in impotence, such a state, whether known or unknown to the parties, makes the promise voidable.

In a state like New Jersey, where a statute makes the marriage of an impotent person void, such a promise is absolutely void (41 N. J. L., 13). In the case of wills, if the testator is un-

conscious of the nature of his acts or incapable of resisting the influence of others, it avoids the will (3 D. R. Pa., 534).

In Torts, drunkenness is no defence in mitigation of damages (68 Ga. 612).

Courts of Equity decline to interfere in favor of parties pleading intoxication in performance of some civil act, but have not gone to the length of enforcing agreements against such parties (57 N. W. Rep., 478). Insurance policies are special forms of contracts, and in life insurance policies, if inebriety is proved as a fact, and the party at the time of filling in the application states he is temperate, such established fact will usually avoid the policy; although a jury has even gone to the extreme of holding that a person may have an attack of delirium tremens and yet be of temperate habits (15 Otto, 350). Suicide, in most life and accident policies, is considered a valid defense against the payment of the claim (190 U. S., 121).

In the case of dipsomania, the party applying for a policy may not know that this is a form of insanity, and if he states merely as his opinion that he is temperate, such will not avoid the policy; but if he states it as a fact, it will avoid the policy (9 App. Cas. 686).

In any case of doubt, however, the question of habits must go to the jury (120 N. Y., 237). as inebriety is regarded as a habit and not a disease in the law on insurance.

It will thus be seen that where the condition of inebriety exists to the extent that the patient may commit offenses against the law, become a victim of designing individuals, unable to conserve his estate, or may become an economic burden upon the State or his family, he should be placed in either a private or public institution for the care and proper treatment of inebriety, voluntarily if possible, but if necessary under legal commitment. New York State recognizes no legal commitment of an inebriate, excepting on the basis of actual insanity, and then only for the period covering the active manifestations of such insanity, so that the few who are actually legally committed as insane persons, are too soon released in a condition in which they almost invariably relapse into their former state. Fortunately for many of these cases, the adjoining state of Connecticut has wisely enacted laws admitting of either the voluntary commitment of the case by the patient himself or a

more binding legal commitment upon the petition of a relative or friend, regardless of the wishes of the patient. In the former case, the patient signs an application for care and treatment in a licensed sanitarium. Should the patient subsequently desire to leave before he is in a suitable condition, he must give three days notice to the physician in charge of the institution. During these three days a relative or friend can make application to have the patient legally committed, against the latter's will, if necessary. The nearest District Court receives the petition, and the judge appoints two examining physicians who examine the patient, and if they advise his further retention, a day is set for the hearing of the case when the patient is brought to court (or his presence may be waived at the discretion of the judge), and if the judge deems it for the best interest of the patient, he grants the petition committing the patient for one year, which can be renewed for another year if necessary. Such a proceeding can be instituted at once by a friend, or relative, without going through the formality of a voluntary commitment by the patient, and this latter is the usual procedure, as few patients are willing to give up the habit of their own free will and accord. Such a patient may be discharged before the completion of the year if fully recovered or the petitioner desires the removal to some other place. This gives the physician-in-charge of such an institution full legal authority to use the necessary legal restraint, to control and prevent the obtaining of alcoholic liquors from without, and direct the habits of the patient; see that he observes regular hours, has suitable diet, proper rest, exercise, recreation, occupation and sleep, together with hydrotherapeutic, electrical or medicinal treatment, according to the requirements of the individual case. Under such treatment as carried out at the present time, from 30 to 50% of inebriates are cured. A still greater percentage of the earlier cases would be absolutely cured and upon their discharge become useful citizens for the remaining years of their life, could we but have an inebriate colony established along similar lines (but especially adapted for inebriates) as that now so successfully carried out for epileptics for the past 46 years at Bielefeld, Germany, where over 2000 cases of epilepsy are under constant supervision; and in the State of New York at Sonyea, Livingston Co., and known as the Craig Colony for Epileptics,

which has been in successful operation for 16 years, and now cares for some thousand patients on a tract of about 2000 acres, on which are situated 76 separate buildings. Such a colony for inebriates should be under State supervision and consist, preferably, of several thousand acres of well-drained farm and woodland in a healthy and accessible location. A detached cottage plan should be adopted with necessary administration buildings, hospital, amusement hall, churches, work shops, dormitories, and numerous detached cottages to accomodate not exceeding 30 persons each. A suitable laboratory for scientific research in the field of inebriety would be of value; also a library for the use of the patients, and staff. Athletic grounds and a gymnasium should be added.

The ground not used for buildings could be cultivated as farm land for vegetable gardens, orchards, poultry and stock yards, and the forest land could be utilized to supply necessary wood. All the work about the colony so far as possible, as assistant nurses, clerks, farm helpers, gardners, etc., could be done by the inmates under the direction of a comparatively small number of paid employees.

It is needless to say that such a colony should be under the direct charge of a medical superintendent with a sufficiently large medical staff, all of whom should be trained inebriatists, or specialists in inebriety, so that each patient might receive individual treatment and be carefully classified. Such a colony could be partially self-supporting, as has been demonstrated at the Craig Colony for Epileptics where, in 1906, the produce from the farm and garden and their numerous industries reached the total value of $42,000.

Finally, the inebriate should be legally committed to such a colony along the lines that have been in active and successful operation in the State of Connecticut for so many years.

The establishment of such colonies upon broad scientific lines, would go far towards solving this serious problem which is becoming of ever-increasing importance, not only from the medico-legal standpoint, but from the sociological and economic standpoint as well. A few statistics from the recent work of Cullen (1907) will illustrate this latter point. The British Nation spends annually in drink £179,000,000. In the United States, in 1905, $1,325,439,074 was spent for the same purpose,

and it was estimated that 1,500,000 men and women were daily incapacitated for work, making a total cost of over $3,000,000,000 annually for and from the direct effects of alcohol. Furthermore, it is estimated that 51% of all paupers in alms-houses came there through drink, and Warden Roberts, of the New York Almshouse, on Blackwell's Island, estimates that 90% of the 2593 inmates are there through drink. Of the 24,300 patients in Bellevue Hospital in 1900, Dr. Alexander Lambert (1904) found that over 25% went through the alcoholic ward. It is furthermore estimated that 10% of all mortality and 20% of all disease is due to alcoholism.

In conclusion, it would seem that as we have experts for all other diseases, alienists for mental disease and neurologists for nervous disease for example, so should we have expert "inebrietists," or specialists on the subject of inebriety to cope successfully with this broad and far-reaching problem.

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FIBROID DEGENERATION OF THE APPENDIX VERMIFORMIS.

BY ROBERT T. MORRIS, A.M., M.D.
Professor of Surgery at the New York Post-Graduate Medical School and Hospital.

Perhaps the time has come for elaborating the subject in such a way as to allow of classification of four rather separate and distinct kinds of appendicitis:

1. Cases of fibroid degeneration of the appendix.
2. Appendicitis with intrinsic infection.
3. Congestive appendicitis.
4. Appendicitis with extrinsic infection.

This classification is a convenient one for practical use, even though we may not be quite ready to adopt it on a basis of scientific accuracy.

Do these four kinds of appendicitis merge into one another? Fifteen years ago, I believed that Cases 1, 3 and 4 commonly merged into cases 2, but observation with reference to this point has had a tendency to disprove that idea, and to separate the different kinds of appendicitis from each other more and more. I am not unaware of the natural tendency for one to make fanciful classifications in any field of science, as one observes, for instance, in the classification of certain sub-species and varieties in botany and in zoology. Nevertheless, it seems to be safe at the present moment to present facts bearing upon the matter of separation of appendicitis cases into four classes.

Class I.—Cases with fibroid degeneration of the appendix are the most common, and yet the ones most frequently overlooked. Fibroid degeneration of the appendix probably sends a much larger number of patients to the doctor than does any other type of appendix trouble. The diagnosis of the condition is frequently made by some physicians, and is not made at all by most physicians, although in my own experience there are few diseases at the present time which send more patients to the doctor. Fibroid degeneration of the appendix may begin in childhood, but it is seldom productive of symptoms before the adult stage of life has been reached, and it is apparently most troublesome in individuals between the ages of 30 and 50.

Senn first called attention to the subject in an article published under the title of "Appendicitis Obliterans" in the *Journal of the American Medical Association*, March 24, 1894. He does not present a detailed study of the changes taking place in the appendix, nor does he offer an explanation for the symptoms, but states that the most constant symptoms are relapsing acute exacerbations of inflammation of short duration, with persistence of soreness and tenderness in the region of the appendix during the intermissions. He advises removal of appendices which are becoming obliterated, on the ground of the prolonged suffering which must ensue if the cases are left to nature.

In 1902, Ribbert, in his "*Lehrbuch der speciellen Pathologie*" on page 319, describes the appendix as an organ which, by reason of its uselessness, begins at an early period to undergo involution. With a lumen completely pervious in youth, it gradually becomes obliterated, so that after sixty years of age over 50 per cent. of cases show total or partial obliteration. Ribbert believes that it is the gradual effacement of the lumen which is largely responsible for the various forms of inflammation of the organ. My observation leads quite to the contrary conclusion, and it is my belief that with gradual disappearance of the structures which are involved in acute infection processes, the tendency to such processes disappears. Further, an appendix which is undergoing fibroid degeneration seems to remain in a condition of irritation which calls out constant protection against infection, and it is my belief that the patients who suffer most often from the symptoms attending fibroid degeneration of the appendix are really the ones who are best protected against infective invasions of the organ. My conclusion has been disputed by surgeons who called attention to the fact that fibrous tissue was found in appendices which they had removed, and which were at the time the seat of acute intrinsic infection. My answer is, that in this particular class of cases, the fibrous tissue appears to represent scar tissue, resulting from the distinctive inflammation of previous acute attacks of infection. Such, at least, has been the history in my cases frequently enough to allow me still to keep the scarred appendices out of the class of fibroid degeneration of the appendix. The reason for the belief that the two kinds of appen-

dices can be kept apart is this: With fibroid degeneration of the appendix there is gradual replacement of all structures of the appendix, but nerve filaments persist longer than other structures, and these persisting nerve filaments, irritated by the contraction of the hyperplastic connective tissue, cause persistent local and reflex disturbance. The scarred appendices, on the other hand, lose nerve filaments along with other structures, as a result of the general toxic destruction of cells at times of acute infection. Consequently, the scarred appendices do not cause in the intervals between attacks, the set of symptoms which are so persistent and so characteristic in cases of fibroid degeneration of the appendix. I would prefer to believe, with Ribbert[8], that fibroid degeneration of the appendix is a menace and provocative of acute infective attacks, because it would then allow one to advise operation in practically all of these cases. From my present point of view, however, it seems best to advise against operation in most cases of fibroid degeneration of the appendix, provided that the patient can get along comfortably, in cases in which there is no disturbance beyond a sense of discomfort in the region of the appendix. Many people with fibroid degeneration of the appendix apparently do not suffer in any way. A certain number of the patients suffer merely from a sense of discomfort in the appendix region. They are inclined to press upon that region with the hand, or to lean against the edge of a table or chair, and they go to the doctor to ask if they have appendicitis. A very large class of patients includes those who, in addition to a sense of discomfort in the appendix region, have occasional attacks of sharp pain, or who suffer from various disturbances of function of the bowel. It is probable that irritation of nerve filaments in the contracting connective tissue of these appendices causes reflex irritation of the intimate ganglia of the bowel wall (Auerbach's plexus and Meissner's plexus), with consequent derangement of function of secreting and excreting organs. Fibroid degeneration of the appendix is, perhaps, the most common single cause for so-called intestinal dyspepsia. It seems to be at least the most commonly overlooked cause. Fibroid degeneration of the appendix is a potent factor in many cases of neurasthenia. In making this statement, I wish it to be clearly understood that I am perfectly familiar with the complex

FIG. 1.—Symmetrical degeneration.

FIG. 2.—Nodular degeneration.

FIG. 3.—Transverse section of the appendix, showing replacement of the inner coats by connective tissue.

[Morris]

nature of a so-called neurasthenia, and know that it is not often that any one disease or defect stands alone as a causative factor. A single disease or defect commonly enough stands alone as a precipitating factor, however, and it is in this rôle, as that of precipitating factor in neurasthenia, that the appendix undergoing fibroid degeneration plays an important part. Fibroid degeneration of the appendix is the cause for trouble in most of the cases in which the patient goes about from doctor to doctor, trying to obtain a definite diagnosis referable to the appendix, and the different opinions which he obtains are apt to leave him much confused. The most common diagnosis which the patient obtains is "chronic inflammation of the appendix," and the most common advice is perhaps to have the appendix removed, on the ground that acute infection of the appendix may appear at any time. Such a diagnosis is not comprehensive, because it does not include the real cause for the chronic irritation, and such advice is unwarranted, according to my belief at the present moment. Examination of appendices which are undergoing fibroid degeneration shows various degrees of replacement of the normal structures of the appendix by connective tissue, and there is apt to be a rather even replacement of appendix tissues, beginning at the distal end and involving lymphoid and submucous tissues primarily. The mucous coat disappears next, then the muscular coat and in some cases the peritoneal coat in part or as a whole. The larger blood vessels of the appendix are pinched out at an early date by the contracting connective tissue, but nerve filaments seem to resist pressure atrophy for a long time. Microscopically these nerve filaments are found to be surrounded by such an abundance of new cells that we have clear evidence of the irritation caused by such appendices. Instead of an even and symmetrical fibroid degeneration of the appendix, we sometimes have localized areas of degeneration representing nodes and internodes, and the internodes sometimes form sites for mucous inclusion. Many years are required for completion of the changes which occur with fibroid degeneration of the appendix, and in the interval some patients will suffer enough to make it worth while to have the appendix removed, because of the influence of that organ upon the general health of the individual, rather than because of danger from infection of the appendix.

The diagnosis of fibroid degeneration of the appendix can usually be made without much difficulty. First, by palpation. If one has acquired a good method for palpation of the appendix, he will have little difficulty in finding these fibroid appendices as a rule. The appendix feels distinctly harder than normal as it slips out from beneath the examining fingers. Tenderness of the appendix may not be a very marked feature, and it is rather less in degree than one expects to find in connection with the history of discomfort, pain or reflex disturbances. The chief diagnostic point in these cases is a very definite one, if taken in connection with accurate palpation of the appendix. The chief diagnostic point consists in finding a point of tenderness at the site of the right lumbar ganglia. For practical purposes one can find this spot by pressing upon the abdominal wall at a point about an inch and a half to the right of the navel, carrying the fingers well down toward the right side of the spinal column. If we find hyperesthesia at the site of the right lumbar ganglia alone, we can assume that the appendix is responsible for this irritation. If, however, the left lumbar ganglia are equally sensitive with the right lumbar ganglia, we must look to the pelvis for the source of the irritation. If neither right nor left lumbar ganglia are hypersensitive, we must look to some part of the abdomen cephelad from the level of the navel. It is when the right lumbar ganglia alone are sensitive that we are to look to the appendix as the single cause for disturbance, as a rule.

In calling attention to the importance of the inflammation which accompanies fibroid degeneration of the appendix, I purposely leave out descriptions of the other types of appendicitis, excepting to state briefly their nature.

Class 2.—Infective appendicitis I believe to be due to invasion of anemic areas in the appendix by bowel bacteria, the anemia usually being a compression anemia, and caused by swelling of the inner structures of the appendix, within a tight outer sheath. The cause for the swelling, whether due to extension of a colitis, the presence of a concretion, or any common cause, I hold to be of small consequence so long as tissues rapidly swell to the point where they suffer compression anemia, with consequent loss of protection from phagocytes.

Class 3.—Congestive appendicitis seems to be a benign con-

dition occurring with congestion of other abdominal organs, and even though the appendix is sometimes distended with serous infiltration to the point of becoming anemic, the congestive process takes place so slowly that protective factors seem to be called out, and there is little danger of these cases merging into Class 2. We find this type of appendix commonly associated with loose kidney, or in cases with abdominal ascites, for example.

Class 4.—Appendicitis due to extrinsic infection is found in cases where such diseases as tuberculosis or cancer involve the appendix. In these cases also, we seem to have protection called out during the slow advance of the disease in such a way as to guard against attacks of acute infective appendicitis proper.

In conclusion, I will repeat that appendicitis may practically be classified in four types, and that fibroid degeneration of the appendix presents the most common and on the whole the most important affection of the appendix, notwithstanding the fact that the phenomena of acute infective appendicitis just at present engage the attention of the profession to a greater degree. It is my belief further, that fibroid degeneration of the appendix protects the patient against acute infective appendicitis, and that it accomplishes this in two ways. First, by removing the structures which would be chiefly engaged during acute infection processes, and second, by the persistent cell protection called out by irritated nerve filaments which are entrapped in the contracting connective tissue.

THROMBO-PHLEBITIS OF THE SIGMOID SINUS AND DEEP JUGULAR VEIN, FOLLOWING MASTOIDITIS—TWO ILLUSTRATIVE CASES.

BY WILLY MEYER, M.D.

Professor of Surgery at the New York Post-Graduate Medical School and Hospital.

Although the operation for mastoiditis and its complications has had to be conceded to the otologist, it nevertheless occasionally happens that the general surgeon is called upon to perform this work. Thus, in the course of many years, it has been my good, or hard luck, to have seen in consultation two serious cases of sinus phlebitis and thrombosis of the deep jugular vein demanding prompt action.

While meditating as to what a general surgeon could best contribute to a Festschrift, dedicated to a colleague who did so much for the advancement of otology, and enjoyed an international reputation among ophthalmologists as well as otologists, it occurred to me that it might not be amiss to present the histories of these two interesting cases.

Case I.—Male, aged 16 years, came under my observation in February, 1899. He gave a history of trouble with the right ear that had existed for a year when, suddenly, septic symptoms developed. An aural surgeon, Dr. M. Toeplitz of this city, was called in. On trephining the right mastoid and finding the antrum filled with pus, he continued the wound down to the dura mater and exposed the sinus. At this point the patient became so weak that the operation had to be discontinued. A few days later he had a sudden chill and developed an effusion into the sheath of the tendons of the right peronei muscles, which was the cause of my being consulted. An incision was made over the swelling and it was found that the sheath contained turbid serum. This was evacuated and drained, but the patient did not improve and, subsequently, he developed a marked swelling on the right side of the neck, causing a good deal of pain. When I was called in again to see the case, it was evident that the sinus phlebitis had extended downward and produced a thrombosis of the deep jugular vein. Of course, operation was promptly decided upon. First the sinus within

the old wound was laid open, and then the internal jugular vein exposed; the latter was found to be much infiltrated and, upon incising it, pus escaped; following its course downward, it proved to be occluded by a thrombus which reached down to the spot where the common facial enters the jugular vein. It was most interesting to observe how, in the facial vein and the central portion of the jugular, circulation was maintained, the tip of the thrombus being constantly washed by the lateral current. It was evident that any moment infectious material could be carried off, which, after passing through the heart, was bound to produce septic embolic pneumonia with pleurisy. Hence, the jugular vein was ligated below this place and extirpated up to the jugular foramen, from which the but slightly adherent thrombus could be removed by means of forceps. The sigmoid sinus having been exposed before, it was now easy to irrigate the intra-cranial part of the vein and thus clear the entire tract of infectious material. The large wound was tamponed and allowed to heal from the bottom.

The patient made a good recovery from the operation, but three weeks later he developed an effusion into both pleural cavities, with symptoms of embolic pneumonia and accompanying pyemic fever. It was apparent that, in spite of the ligature of the vein within a healthy area, the inflammation had progressed, thus producing this alarming complication. The pleural cavities were tapped several times and considerable turbid serum withdrawn. However, after weeks of serious illness and under careful nursing, the patient recovered, there remaining a large cavity in the region of the right mastoid, which, in July, 1900, necessitated further operating. The bone was laid open and a granulating mastoiditis found; the wound was deepened as far as disease was discernible, but the antrum was not reopened. Later, the granulating surface was covered with Thiersch's grafts, all of which took nicely. About a month later the upper part of the grafts gave way and a sinus again appeared. In this condition the patient was seen by a number of ear specialists until, finally, he came again under my care, and, with the assistance of Dr. Toeplitz, the regular Stacke operation on the mastoid was done; the same proved entirely successful. The patient completely cured, could be presented before the N. Y. Surgical Society on April 24, 1901.

This case represents recovery after true pyemia following purulent otitis media with sinus thrombosis and consecutive inflammatory involvement of the deep jugular vein.

Case II.—Female, 34 years of age, with chronic otitis media, not responding to ordinary treatment. On May 7, 1903, a well-known otologist performed a mastoid operation, which, though done with all the usual care, was followed by severe local inflammation with general manifestations. Slowly an infiltration developed on the corresponding side of the neck.

When seen by me in consultation on May 22, 1903, there was edema and tenderness of the neck and face, evidently due to a suppurative phlebitis. Operation done the following day, proved the deep jugular vein to be completely thrombosed. The vein was carefully followed downward toward its junction with the subclavian; but the thrombus was found to extend beyond such part of the trunk as could be made accessible; it evidently reached down to the very junction of jugular and subclavian. It was not deemed advisable to resect the clavicle in order to gain better access. Of course, it was very tempting, after the vein had been split as far downward as was possible, to pull the thrombus out of the lowest portion of the jugular and then, after sufficient hemorrhage had washed out the parts, to place the ligature. However, there were quite some adhesions between venous wall and thrombus, which latter, in this region, did not show any suppurative foci; besides, the danger of some small particle being carried off by the strong blood current of the subclavian, and then causing mischief in the lungs, was considered. The lower part of the vein was, therefore, left undisturbed, while the upper section was thoroughly cleared of all débris, but not excised, as the patient was weak and the operation had to be finished as quickly as possible. The entire wound was tamponed with aseptic gauze, which was kept moist. The patient made a slow recovery, without any further complications. She is in perfect health to-day.

Reviewing these two cases, it will be seen that extirpation of the deep jugular vein, as well as mere incision with thorough removal of the thrombus and drainage, can effect a permanent cure.

Although it would be almost presumptuous to draw any conclusions from these two cases, I personally would, never-

theless, feel inclined to state, that excision, though desirable, is not absolutely necessary. Thorough cleaning of the large viscus of the vein may suffice, in many instances, and, perhaps, cause less disturbance in the system, than the loosening and excision of the trunk with the necessary ligation of the tributaries. It seems to me that the decision as to which course to pursue in the given case, should be made to depend largely upon the condition of the patient.

When called upon to operate on these seriously sick patients, the thought suggests itself whether these complications, at least in the acute cases, could not be avoided by prophylactic measures, and in a sufficiently large proportion of cases to warrant our advising such prophylactic treatment as a routine measure.

The prophylactic treatment I have in mind, in this connection, is Bier's hyperemic treatment, which in cases of acute otitis media, with or without mastoiditis, has already had great triumphs. As in general surgery, medicine and other branches of medical science, here, too, Bier has been the path-finder. With the help of the well-known otologist, Professor Eschweiler of Bonn, he has proved beyond the shadow of a doubt, that by the prompt application of obstructive hyperemia of the head by means of the elastic neck band, immediately after the onset of the symptoms of otitis media, the disease may often be controlled and further complications avoided.

If an abscess has already formed over the mastoid, this is incised. But instead of draining it, suction treatment is applied in addition to the neck constriction.

The method is equally applicable to children. Here, too, the comfortable feeling of the little patients must be the index to the degree of constriction, to be employed. If they breathe, eat, drink and sleep well, we may know that the treatment is correctly applied. It is a frequent occurrence that the older children actually clamor for the reapplication of the bandage, as the headaches begin to return during the two hours in which the bandage is removed each day.

Naturally, if examination of the drum membrane points to the presence of pus within the middle ear, paracentesis of the drum membrane must precede hyperemic treatment, one of the fundamental rules in the carrying-out of Bier's treatment being, that

whenever an abscess is diagnosticated, it should be primarily incised.

Careful observation is necessary, in order to determine the indication for mastoid operation. If such careful observation can not be bestowed upon the patient in private practice, he had better be promptly admitted to a hospital. In the event of the treatment being successful, the clinical picture changes rapidly; the fever subsides, the pus discharge diminishes and a general euphoria soon sets in, so that it is difficult to keep the patients in bed.

Bier's personal results with the method at the Bonn clinic, shortly before his removal to Berlin, are absolutely convincing, and the facts, established by him, remain unchanged, despite the almost universal condemnation of the procedure at the Congress of Otologists that met in Vienna in October, 1906. The advantages of the method are so manifest, that it will be impossible for otologists to permanently close their eyes and ears to the truth of Bier's teachings.

The experience of operating upon the mastoid with the positive expectation of encountering pus, only to find the cavity empty, showing that the operation might have been avoided, is not foreign even to the most expert of specialists. I believe that if in these cases the time of observation, before operation is decided upon, were utilized for a careful test of artificial hyperemia, operation might often become unnecessary.

It is gratifying to note that otologists here have lost no time in taking up and following Bier's suggestions. The excellent paper by Kopetzky, assistant surgeon to the Ear Dispensary of the Manhattan Eye, Ear and Throat Hospital, read before the section on otology of the N. Y. Academy of Medicine, on April 12, 1906, amply proves this. It is here shown that six out of seven cases of acute otitis media purulenta with acute mastoiditis, and one case of distinct sub-periostal abscess, in the service of Dr. Wendell C. Phillips, professor of otology at the N. Y. Post-Graduate School and Hospital, were completely cured by hyperemic treatment of an average duration of five to six days. Just as in Bier's own cases, the ear function was restored much earlier than is generally the case under the usual treatment.

It is not claimed by Bier or any of his followers, that every

case treated early in this way can be brought to healing without operation. Artificial hyperemia is not a panacea; but the successes already obtained with the method in these cases are such as should make it incumbent upon every thinking otologist to investigate and test for himself the truth of the claims made for it. Certainly, it must be welcome news to learn that a means has been discovered, that will aid him in his efforts at combating a disease that is so frequently followed by some of the most dreaded sequelæ, *i.e.*, sinus phlebitis, brain abscess and meningitis.

We must not lose sight of the well-known axiom that *prophylaxis*—properly directed work, in order to prevent the appearance or development of a threatening disease—is the highest aim of medical science.

BLADDER WOUNDS IN HERNIA OPERATIONS.

BY W. B. DE GARMO, M.D.
Professor of Special Surgery (Hernia), New York Post-Graduate Medical School and Hospital.

For several centuries it has been known that the bladder sometimes enters as a factor into the formation of abdominal hernia; and its presence has been recognized as constituting a complication in the operative treatment. At rare intervals this fact has been mentioned in the literature of the subject, but the average surgeon either overlooks, or promptly forgets, the existence of this occasional complication.

The term " cystocele " has been applied to hernias containing the bladder, but the use of this word is undesirable and misleading, as the gynecologists have now appropriated the term and applied it to a condition of bladder prolapse into the vagina.

During the past 20 years the operation for the radical cure of hernia has become so general, and consequently the number of accidental openings of the bladder so common, that it has become an object of close study with every surgeon how best to avoid such an unpleasant, and sometimes disastrous, accident. Yet, even with the greatest care, such an accident is pretty sure to happen to almost every man who operates extensively for hernia, and he will discover, to his great chagrin, that he has unknowingly cut into the bladder under the supposition that he was opening a hernial sac, a cyst, or hydrocele of the cord; or he will find that the loose mass of fat that he has tied off and cut away was, in reality, a diverticulum or elongated part of the bladder wall.

In my own experience of 21 cases of hernia containing the bladder, I was able to recognize and avoid accidental injury to that organ in all but one case. My carefulness was prompted by witnessing early the accidental opening of the bladder by a surgeon who was known the world over as a careful, conservative operator; and upon this occasion I resolved, that if this error could be avoided by care, I would never fall into it. For many years afterwards this caution served me well, but the time came when my pride had a fall — and great was the fall thereof, because I committed my error while operating upon

the person of a well-known physician for whom I had the most profound respect. In this one lapse I opened promptly, and without hesitation, what I believed an ordinary hernial sac, but which I found to my extreme mortification was not a hernial sac but the bladder.

Dr. B. Farquhar Curtis[1] of New York was a pioneer in this country in compiling the cases in the literature of this subject. From the earliest times to the date of this paper (1895), he found 58 cases recorded, and in 41 of them the bladder was wounded during the hernial operation. Only two years after the paper of Curtis, Dr. C. L. Gibson[2] tabulated 45 additional cases, which with three exceptions, had been reported in that interval. In 16 of these 45 cases the bladder was injured during operation, and the mortality was 12½%.

The increasing importance of the subject is shown from Gibson's paper in which he tabulates 45 cases covering the period 1895–7, while Curtis found only 58 cases in the whole previous literature covering a period of 320 years. From 1897 to 1908 there are recorded some 300 additional cases.

Etiology.—The bladder complication of hernia occurs in the sexes in about the same proportion as usually found in the ordinary types. In the 103 cases of Curtis and Gibson just enumerated, 77 were inguinals, and 22 femoral hernias. Of the former 70 occurred in males and seven in females; and of the latter six were in males and 16 in females.

The youngest case on record is that of Adams,[7] who reports a bladder complication in an 18 months' old male infant. In my cases I find, however, that fat men over 50 years of age are particularly prone to the bladder complication, as are also those who suffer from recurrent hernia; for the latter the operative procedure of pulling down the peritoneum when tying the sac, is to blame. The top of the bladder is dragged towards the inguinal region and is placed in a favorable position to protrude with subsequent hernia.

Anatomical Considerations.—On reviewing the anatomical relation and position of the bladder in the pelvis, one is not surprised that this organ should occasionally protrude as a hernia, with or without accompanying abdominal viscera. (Fig. 1.)

When empty, the bladder lies wholly within the pelvis and the peritoneum covers all portions of its surface, except where

it lies in contact with the pubic bones, triangular ligament and lower part of rectum, or cervix uteri and vagina (*Deaver's Surgical Anatomy*).

When distended the bladder may reach as high as the umbilicus behind the recti muscles, and the area covered by peritoneum is then mostly posterior and is bounded above by the urachus and below by the line connecting the orifices of the ureters and at the front of the sides by the obliterated hypogastric arteries. The anterior and lateral sides of the organ, which are not covered with peritoneum, are loosely united to

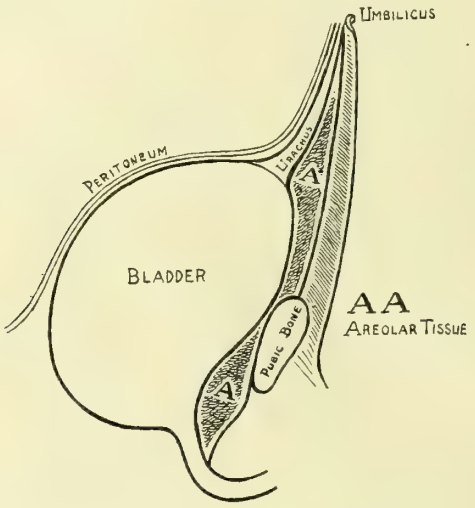

Fig. 1.—Schematic illustration of the position and relation of the moderately distended bladder.

the adjacent structures by areolar and fatty tissues containing numerous veins. This prevesical areolar tissue and fat is continuous with the extra-peritoneal areolar tissue.

Distention and movements of the bladder are favored by these loose connections and it is not surprising that the distended organ, under the downward pressure of a strain, should follow the direction of least resistance, and prolapse as a direct hernia through the extra-peritoneal areolar tissue to the inguinal canal near the external abdominal ring. (Fig. 2.)

In infancy and childhood these conditions are unlikely as

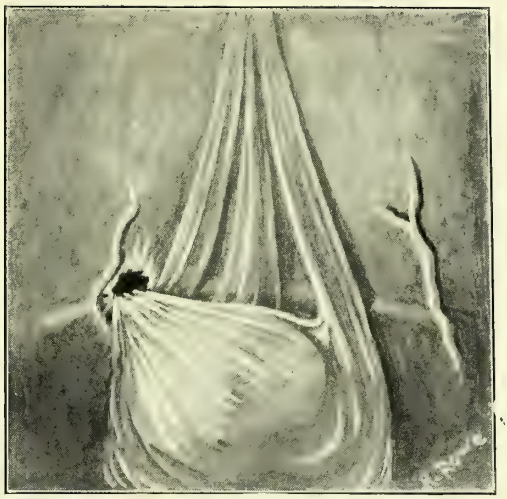

Fig. 2.—Abdominal wall from within, showing top of the bladder dragged into the hernial opening by the peritoneum between the epigastric artery and the obliterated hypogastric artery.

the organ projects well into the hypogastric region and is also much narrower than in the adult.

The normal bladder wall in section shows fibrous, muscular, submucous and mucous coats. When moderately stretched it is $\frac{1}{8}$ of an inch thick, and the mucous lining appears to the eye as a pale rose color. When the bladder is well distended, however, as may result from chronic obstruction to the exit of urine, the wall thins so materially that these layers cannot be made out, and sacculi of the thinned mucous lining appear on the surface of the bladder, having protruded between the attenuated bands of the muscular coat.

One surgeon operating for hernia, saw fluid through this membrane, and as the sacculus was near the spermatic cord, opened it for hydrocele of the cord. Others report they were unable to recognize the bladder even after opening into it, so changed in appearance had the diverticulum become.

Cause and Formation.—From the anatomical data just reviewed, some of the causes of hernial prolapse of the bladder are quite evident: (1) the position of the viscus behind the pubic bones, which, in distending, broadens towards the inguinal and femoral openings; (2) the distribution of loose areolar tissue and fat anteriorly and in front of the sides of the bladder up to the obliterated hypogastric arteries, and, while the recti muscles prevent any prolapse of the organ in front, the continuation of this areolar tissue to that of the extra-peritoneal areolar tissue, allows a diverticulum of the bladder to dissect down into the triangle below the border of the internal oblique to the inguinal canal. Other causes are: muscular strain on a distended bladder, and constipation. The latter acts as a factor in two ways: first, by the rectum, over-distended, lifting the bladder out of the pelvis, and secondly, inordinate straining at stool.

The attachment of the peritoneum to the bladder is responsible in some cases of large hernias. The peritoneum is pushed down with the descent of the hernial sac to such an extent that it drags the bladder down also. Operators accomplish the same feat by overzealously pulling down the sac in its resection. The records show a number of cases in which the bladder was wounded or tied into the neck of the sac in this way.

These bladder protrusions are classified as regards the presence

or absence of that part of the bladder which is covered with peritoneum.

A. Extra-peritoneal: protrusion through one of the hernial openings of that part of the bladder wall covered only with areolar tissue.

B. Intra-peritoneal: peritoneal covered bladder wall is dragged down into the sac by the preceding abdominal viscera.

C. Extra and intra-peritoneal: in which both types occur.

Two-thirds of all cases are of Class A; the extra-peritoneal type.

Preoperative Diagnosis.—Rarely has the diagnosis of the bladder complication in hernia been made positively before operation. Certain symptoms associated with the voiding of urine, or the position and character of a tumor above the pubic bone in the direct type of hernia, may suggest such a complication to the operator and put him on his guard. In the 171 cases collected by Brunner (quoted by Moynihan[4]), in only 19 of them (or 11%) was the bladder complication diagnosed before operation. Karewski[19] reports five cases of bladder hernia, in two of which he was able to make a positive diagnosis with the aid of the cystoscope.

There are certain symptoms, as suggested, that point towards a diagnosis. A patient may complain of a desire to urinate when pressure is made upon the tumor, and in one of my own cases, an enormous scrotal hernia, the patient declared that he was obliged to compress the hernia to completely void his urine. Another, a physician, suffered great irritability of the bladder during the day (though not at night), which soon disappeared when the hernia was supported by a truss.

Some characteristics of the tumor also help in diagnosis. For example, bladder hernias, even of large size, show little tendency to drop down into the scrotum. They reduce slowly and incompletely, and are quite liable to become painful under truss wearing. However, it must not be lost sight of that, even in extreme cases of bladder involvement, no symptoms pointing towards that organ may occur.

Diagnosis at Operation.—Having these suggestive symptoms in mind no surgeon should be wholly unprepared. Further data at the operation should assist him in recognizing the bladder, if he interpret them intelligently. A hernial sac that

comes out immediately above the pubic bone into the inguinal canal, on the inner side, or behind, the cord, may well be bladder. If this same tumor is covered with fat of a light lemon color (prevesical fat), from which it separates with difficulty and does not "shell out," the diagnosis should be positive. Again, a bladder diverticulum is often attached to the surrounding tissues by adhesions. It is useless and misleading to watch for normal bladder wall with muscular fibres, as it becomes so thinned and changed in character as to be unrecognizable.

A sound passed into the bladder through the urethra at this junction and felt at the tumor wall, would of course be conclusive evidence.

The one procedure that has proven of the greatest service

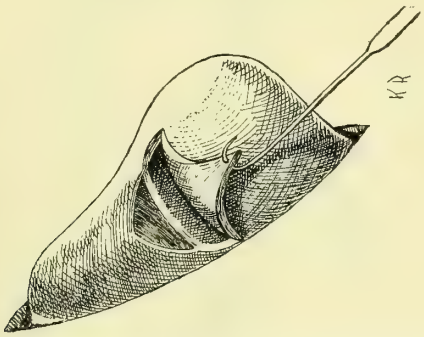

FIG. 3.—Common form of protrusion where both peritoneum and bladder wall are protruding. Section shows dividing line. Incision above this line enters the peritoneal cavity; below, it opens into the bladder.

to me is to open into the abdominal cavity high up in the canal and examine the tumor *from the inside.* In some cases two sacs may present on the inner side of the inguinal canal. (Fig. 3.) By opening the upper one near its neck and passing the finger into the abdominal cavity, the lower sac may be identified and treated accordingly.

In instances where the bladder is opened or wounded at operation, the appearance of the mucosa of bladder, when recognizable, or the presence of urine, are further evidence to the above in establishing its identity.

However, notwithstanding all these points of diagnosis, the literature shows many cases in which the bladder hernia was not recognized even on opening into it. In one case,

a sacculus of the bladder wall was ligated with the hernial sac and cut away. In others the viscus was injured by picking up and ligating what appeared to be a mass of loose fat deep in the canal. And in still another, the ureter, as well as a part of the bladder wall, was ligated and cut away. The accidents in these cases were only realized when urine leaked through the inguinal wound, or blood was present in the voided urine.

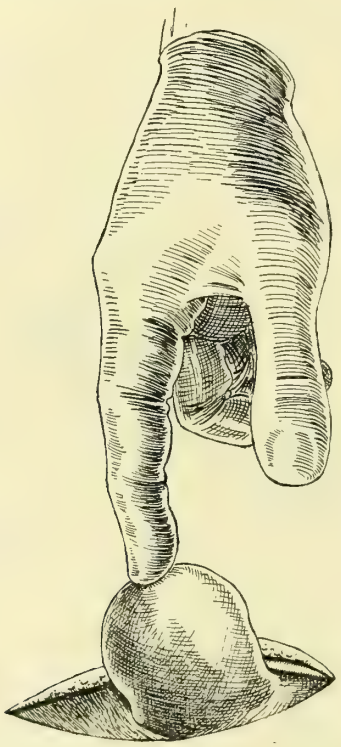

FIG. 4.—Diverticulum, or protrusion of the bladder wall, about to be inverted.

In two cases the autopsy showed that the patient had died from shock due to the extravasation of urine into the peritoneal cavity and loss of blood from an unseen wound.

Management of Bladder Complication in Hernia.—The special operative procedure necessary is to remove the bladder protrusion or diverticulum as far from the immediate vicinity of the canal as possible, otherwise, the hernia is quite liable to recur.

Personally, I have resorted to two methods. In the first, the bladder hernia is freed from the surrounding tissues by blunt dissection and carried well towards the median line, and held there by suturing the prevesical fatty fascia to the conjoined tendon. Adhesions forming, the viscus is held quite satisfactorily. By passing the blunt end of the needle carefully through the prevesical fascia, there is little danger of puncturing the wall of the bladder.

My preference is strongly for the second method, which I call the method of "inversion." A purse-string suture of

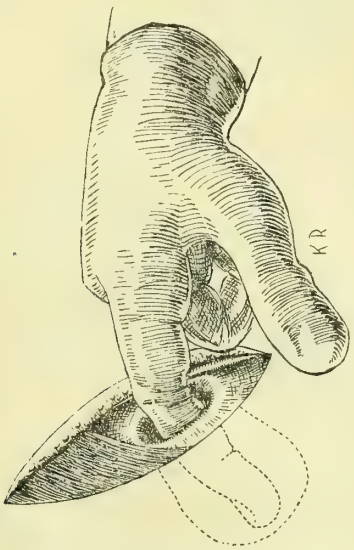

FIG. 5.—Diverticulum inverted into the bladder.

catgut is run around the neck or the border of the protruding diverticulum, or sacculus, which is then completely inverted, without opening, back into the bladder, just as the cut appendix is invaginated into the cæcum, and the purse-string suture securely drawn tight and tied. (Figs. 4, 5, 6.) The same precaution is taken in placing the purse-string suture in the muscular coat of the bladder wall, as is followed in running that suture in the cæcum in the operation of appendectomy. The illustrations convey an idea of the method more clearly than words. The first method should be confined to those cases in

which the protruding tumor sac is large and sessile, for without a neck the second procedure is impracticable.

For the bladder wounds which have caused such a high mortality, a number of methods of closure are advocated. Dr. Howard A. Kelley* advocates catgut sutures through the entire thickness of the bladder, reinforced with a second row of fine silk sutures. He says: " I have experienced no trouble in tying catgut sutures in the bladder, and dropping them when it was easier to pass and tie the sutures at the base." In Dr. Wier's case, reported by Gibson, continuous catgut sutures were used in apposing the mucosa, and interrupted Lembert sutures of

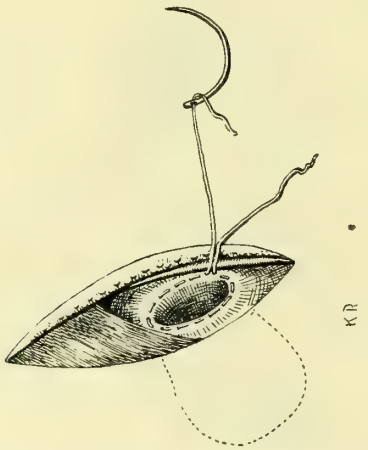

FIG. 6.—Suture in place to close the opening after inversion is complete.

fine silk for the second tier. A catheter was kept in the urethra for three days.

Chas. H. Mayo closes the bladder wounds with catgut protected on the outside by linen sutures of the Cushing type. Gibson advises tier sutures, with preference for silk, the bladder drained per urethra by a catheter for a few days in the intra-peritoneal wounds, and gauze packing to the bladder wound until its healing is assured. In my own case (see case histories) the silk sutures proved such an annoyance that I feel it would be better to take the risk of leakage from the too early absorption of

* " Resections of the Bladder in Rebellious Cystitis," *N. Y. State Jour. of Med.*, April, 1906, p. 145.

catgut, than to leave the unabsorbable silk to protrude finally into the bladder.

So it seems most rational to me to stitch up the rent or incision in the bladder by continuous catgut sutures for the apposition of the mucosa, followed by one or two layers of sutures through the muscular and fibrous coats, of silk or linen Lembert sutures, interrupted or continuous. When the bladder wall is very thin, one must infold one layer and close the other over it. The bladder can best be kept empty by frequent catheterization rather than leave the catheter continuously in the urethra; and it is by all means best to close the abdominal wound to ensure the cure of the hernia by primary healing of the tissue, and resort to packing with gauze only as a last resort in extreme cases.

HISTORIES OF PERSONAL CASES.

Case I.—Intra-peritoneal (Card No. 121), December 4, 1895. T. S., age 50 years. Right scrotal hernia of gradual growth and 20 years' duration; no previous treatment; tumor two feet in circumference, extending 14 inches below the external ring; no symptoms of strangulation; incontinence of urine at times. When urinating the patient is frequently obliged to compress the tumor quite violently with both hands in order to complete the act. Previous venereal infection or genito-urinary trouble was denied.

Operation was performed at the Post-Graduate Hospital, Dr. Ogden and staff assisting. The sac was found to contain large and small intestine, omentum, and a mass protruding with peritoneal covering, believed to be the bladder, all freely reducible. The entire sac was stripped out, tied at its neck with kangaroo tendon, and cut away. The canal was closed by the Bassini method, kangaroo tendon being used as suture material. On the following day the house surgeon reported complete retention of urine and inability to pass the catheter. The writer being out of town, the case was seen by Dr. George E. Doty, who found the bladder enormously distended, almost to the umbilical line, and tapped it supra-pubically with an ordinary trocar. On the second day, Professor Eugene Fuller opened the bladder by perineal section, leaving liberal drainage.

The case made an uneventful recovery, the temperature at

no time exceeding 99½ and the hernial wound healing by absolute primary union. There has been no recurrence of the hernia.

Case II.—Extra-peritoneal (Card No. 476), January 5, 1899. C. D., large fat man, 58 years of age. Left scrotal hernia larger than adult fist, of 30 years' duration, and not fully reducible; trusses worn found painful and unsuccessful; condition gradually growing worse; repeated attacks of strangulation and inflammation.

Operation was performed at the Post-Graduate Hospital, Dr. McAlpin and staff assisting. On opening the canal two distinct sacs were disclosed, one coming down the canal from the internal ring and about three inches in length. When this was separated from the cord and raised up, another sac of direct hernia type was found protruding on the inside of the cord close to the pubic bone. This lower protrusion appeared like a thick mass of fat well supplied by vessels and connecting with the deeper parts by a broad base. Bladder tissue was suspected, and a sound introduced through the urethra passed readily into this diverticulum. After ligating and removing the upper sac, the lower mass was separated from its surrounding with considerable difficulty and the canal closed over it by the Bassini method. It was thought that considerable trouble would result from the necessary handling and tearing of tissue, but there was prompt recovery without anything more unfavorable than a slight superficial suppuration. No recurrence five years later.

Case III.—Extra-peritoneal (Card No. 496), April 12, 1899. Rev. J. S., a thin man, age 71 years (referred by Dr. L. Bolton Bangs). Double inguinal hernia for many years; the size of a hen's egg on the right and as large as a fist on the left side; truss-wearing painful and hernias not retained; twice strangulated; complained of malignant growth.

Operation was performed at the Murray Hill Sanitarium, Dr. George E. Doty assisting. On the left side a large sac, difficult to remove, was found, but without other complications. On opening the canal on the right side a tumor, the size of a small hen's egg, was also found protruding inside of the cord and immediately above the pubic bone. Its neck was rather broader than usual for a hernial sac, and its anterior surface could easily be followed down to the bladder wall. It was easily separated from its adhesions, inverted and buried under the muscular wall

by the Bassini method of closing the canal. Primary closure was obtained on both sides and the patient left the sanitarium on the 14th day after the operation. Operation was followed by almost complete relief of abdominal symptoms.

Case IV.—Extra-peritoneal (Card No. 534), July 19, 1899. Physician, age 61. Inguinal hernia on the right side, size of English walnut, noticed about three months since. Truss proved irksome and operation desired.

Operation was performed at the patient's home, assisted by Dr. George E. Doty. Canal was found well filled by an elongated piece of fat that came from vicinity of internal ring. The hernia was found to be a direct protrusion inside of the cord, close to pubic bone, and with a broad base. It was thickly covered by fat, closely adherent to the surrounding parts, and easily recognized as an extra-peritoneal protrusion of the bladder wall. It was turned in and the canal closed by the Bassini method. No recurrence to time of his death from other causes four years later.

Case V.—Extra-peritoneal (Card No. 551), August 17, 1899. H. P. W., age 45 (referred by Dr. Barker, Morristown, N. J.). Left inguinal hernia for several years, fairly well controlled by a good truss; truss-wearing always attended by considerable pain.

Operation was performed at the Murray Hill Sanitarium, Dr. George E. Doty assisting. Canal was filled with fat which was ligated and cut away. Tumor, half size of a hen's egg, protruded inside of the cord and by a broad base closely adherent to all surrounding parts. The Anterior surface was found to dip down deep into pelvis close to pubic bone and was recognized as part of bladder. A stitch of catgut was passed through the top of the tumor, carefully avoiding the bladder wall, and it was then carried towards the median line through the conjoined tendon and tied. This was done in order to keep the diverticulum away from the canal. The canal closed by Bassini method, healing by primary union, and patient returned to his home on 14th day. No recurrence to January, 1908.

Case VI.—Para-peritoneal (Card No. 1089), February 11, 1904. J. C., age 38 years, medium build. Right direct hernia, size of goose egg, of several years' duration; truss-wearing painful and abandoned; incarcerated when first seen and reduction impossible, but no symptoms of intestinal obstruction; diagnosis

of incarcerated omentum and patient sent to hospital for operation.

Operation was performed at the Post-Graduate Hospital, Dr. Loughran and staff assisting. On opening the canal a bulging of tissue into its upper part was found, and this merged into a decided tumor protruding inside of the cord close to the pubic bone. Constriction of this tumor had been at the external ring. The amount of fat and the unusual vascularity of the mass caused me to be suspicious at once that it was the bladder. This was verified by opening into perineum high up in the canal. It was easily ascertained from within the abdomen that the larger mass was the bladder protruding extra-peritoneally. It was freed from adhesions, inverted and buried under the muscular wall by the Bassini method. Recovery by primary union without incident.

Case VII.—Extra-peritoneal (Card No. 1138a), May 30, 1904. Physician, medium height and rather stout, about 60 years of age. First seen May 19, 1902. Diagnosis, left complete inguinal hernia, of two months' duration, size of two fingers through the canal; believed to be omentum or fat; supposed cause, cough; truss applied, which controlled fairly well, but irksome and operation desired.

Operation was performed at Dr. Bull's private sanitarium. Dr. George E. Doty assisting. On opening the aponeurosis what appeared to be a fatty sac was found well up in the canal. It came out inside of the cord and connected with the abdomen by a rather broad base, but entered the canal higher up than any bladder protrusions seen previously or since, and presented every appearance of a hernial sac well covered by fat. On opening this, it was found to be a diverticulum from the bladder. Its walls were fatty, not thick, and presented none of the muscular covering supposed to be found in the bladder wall. The incision made in the bladder was about $\frac{3}{4}$ of an inch long. The mucous coat was brought together by small catgut, infolded, and a layer of small intestinal silk used to bring together the outer surfaces. Special care was used not to lacerate the mucous lining of the bladder. Still another row of silk sutures was placed outside of this. The bladder was then pushed back and the muscles closed over as in the Bassini operation. There was no true hernial sac in the canal. Some loose fat was tied off and removed.

A catheter was tied in the bladder and the patient put to bed. The catheter was removed only for cleaning and was kept in place for four days. Aside from some slight irritation from the catheter, recovery was prompt and apparently complete, the patient leaving the sanitarium at the end of two weeks. He was seen at the end of three weeks and then made little complaint regarding his bladder, though he had some irritation on urinating.

The patient went to Cooperstown on June 25, 1904. On the 27th, he developed a chill with temperature of 104°. The temperature subsided in a few days. On July 28 following, he had another chill with temperature 105°.

Dr. Eugene Fuller operated July 30. The prostate was removed and found enlarged and hard. There was no abscess. Dr. H. T. Brooks subsequently reported in the pathological findings that the hardening and enlargement were purely of an inflammatory character. Recovery was slow.

On November 30, 1904, and again on February 14, 1905, a small calculus was crushed and washed out of the bladder. The washings from the bladder on both occasions showed some silk thread.

At the time of the prostate operation, Dr. Fuller, at the suggestion of Dr. A. H. Cilley, who had been present at the hernia operation, examined the interior of bladder at the site of the hernia wound and found that it was all right. There was no silk there then or he would have felt it. The silk must have been extruded into the bladder after that date. Whether it may have been disturbed at the prostatic operation, which went through both bladder walls, it is impossible to say; certain it is that the presence of the silk gave the patient great distress and delay in recovery.

Case VIII.—Para-peritoneal (Card No. 1177), November 30, 1904. W. C., a fat man, 60 years old. Double inguinal hernia, four years on left side and six months on right; truss-wearing painful and not retaining hernia.

Operation was performed at the Murray Hill Sanitarium, Dr. George E. Doty assisting. Left herina of ordinary direct type was found bulging into canal. The bulk of the tumor protruded inside of the cord very low down. I opened into the peritoneal cavity high up and found that the lower swelling was the bladder protruding extra-peritoneal. The split in the peritoneal sac

was stitched up with catgut and the whole mass was buried under the internal oblique muscle by the Bassini method. The hernia on the right side was of the ordinary oblique type. Recovery was prompt with no recurrence.

Case IX.—Para-peritoneal (Card No. 1184), December 15, 1904. Italian, age 38 years. Medium build; double inguinal hernia; previous history unknown.

Operation was performed at the Post-Graduate Hospital, Dr. Parker and staff assisting. On the left an incomplete oblique hernia was found, but on the right the hernia was of the direct type, with peritoneum bulging into the canal. When this was opened it was found that the lower and larger protrusion was bladder wall, extra-peritoneal. The peritoneum was closed by purse-string suture of catgut, and the bladder, after being freed from adhesions, was inverted, and buried beneath the internal oblique muscle by the Bassini method.

Case X.—Extra-peritoneal and hernial sac (Card No. 1192), January 6, 1905. M. K., age 44. Man about five feet two inches high, weighing over 200 pounds, with right inguinal, umbilical and ventral hernia, all of several years' duration.

Operation was performed at the Murray Hill Sanitarium, Dr. George E. Doty assisting. On opening the canal a sac about three inches long was separated from the cord up to the internal ring. This left a swelling as large as half-hen's egg protruding inside of the cord low down in the canal. Upon exploration through the upper sac this was found to be the bladder extra-peritoneal. It was inverted and buried under the internal oblique muscles after ligating the true hernial sac. Ventral and umbilical hernia were operated upon at the same time. Primary union. Returned home on 14th day. No recurrence January, 1908.

Case XI.—Extra-peritoneal and hernial sac (Card No. 1195), January 9, 1905. Rev. W. I. H., 47 years of age, and with strong tendency to adiposity. Double inguinal hernia of many years' duration; on the right side, painful and not retained by truss; on the left, of recent origin and easily controlled.

Operation was performed at the Post-Graduate Hospital, Dr. Finley and staff assisting. On opening the right side the canal was found full of loose fat and bulging peritoneum. Lower in the canal the bulging (hernial sac) merged right into a tumor the size of a small hen's egg, which connected with the abdomen

by a broad base. The case was so similar to the one operated on three days previously (reported above), that the bladder was at once suspected, and it was verified by opening the upper sac and examining the lower tumor from within. The opening in the sac was then closed by purse-string suture of plain catgut. the bladder freed from adhesions, carried towards the median line and the overlying fat fastened there by one suture through the conjoined tendon. The canal was then closed by the Bassini method. On the left side the canal was found full of loose fat, but no sac. The patient left the hospital on the 14th day perfectly healed. No recurrence January, 1908.

Case XII.—Extra-peritoneal (Card No. 1203), February 13, 1905. A. W., age 61 (referred by Dr. S. W. S. Toms, Nyack, N. Y.). Large man of medium build; right inguinal hernia, three years' duration, supposed to contain omentum. A powerful truss worn but failed to retain hernia.

Operation was performed at the Murray Hill Sanitarium, Dr. George E. Doty assisting. On opening the canal a tumor the size of a small hen's egg was found protruding inside of the cord and immediately above the pubic bone. Owing to its vascularity, the bladder was suspected, and on tracing anterior surface of the tumor it was found to dip deep into the pelvic cavity, following closely the inner surface of pubic bone. No hernial sac existed and the abdomen was not opened. The bladder was freed and pushed well towards the centre of the abdomen and the canal closed carefully by the Bassini method. The patient left the sanitarium on the 14th day perfectly healed. The patient had considerable pain for four or five months and hernia recurred about one year later.

Case XIII.—Extra-peritoneal (Card No. 1234), May 2, 1905. F. E., age 38 (referred by Dr. Abrahamson). Thin man of medium height; double inguinal hernia for six months; no treatment.

Operation was performed at the Post-Graduate Hospital. On the right side a tumor the size of an English walnut was found protruding inside of cord, with a very broad base and thickly covered with fat. It was considered suspicious and the peritoneum was opened near the internal ring. The tumor was found to be an extra-peritoneal protrusion of the bladder, which was freed from adhesions, pushed well to centre, after inverting, and the muscles closed by the Bassini method. The opening in the

peritoneum was previously closed by catgut. On the left side a sac very similar in character was found, which was believed to be the bladder, but this was not verified. The procedure was the same as on the opposite side. The patient left the hospital on the 14th day perfectly healed. No recurrence.

Case XIV.—Extra peritoneal (Card No. 1239), May 10, 1905. E. A. T., 53 years of age (referred by Drs. Roberts and Fleming). Patient of medium height and quite fat. Double inguinal hernia of four years' duration; trusses unsatisfactory and worn only part of time.

Operation performed at Murray Hill Sanitarium, Dr. George E. Doty assisting. Right hernia the size of two adult fists, was found full of hypertrophied omentum, which was ligated by chromic gut and cut away. The canal was closed by the Bassini method. On the left side a small oblique sac was found protruding through the internal ring, and lower down inside of the cord, was what appeared to be a direct hernia. Upon examining this through an opening in upper sac,it was found to be an extra-peritoneal bladder protrusion. The bladder was freed and pushed well to the center, the neck of the sac ligated, and canal closed by the Bassini method. Primary union was secured on the left, but slight suppuration occurred on the right. The patient left the sanitarium on the 21st day. No recurrence to January, 1908.

Case XV.—Labial hernia, para-peritoneal bladder protrusion (Card No. 1295), January 3, 1906. K. W., age 36 (referred by Dr. Millspaught). Diagnosis: right labial hernia, the size of an adult fist, containing intestine and omentum; of nine years' duration; some symptoms of strangulated hernia during the past year; no truss worn.

Operation was performed at Murray Hill Sanitarium, Dr. George E. Doty assisting. On opening the canal a large sac was found which was of ordinary thickness in its upper part, but was thick and had the feeling of adherent omentum at its fundus, which was well down in the labia majora. On opening the tissue near the internal ring a large, empty sac was found, which had dragged the bladder well over the pubic bone. On its posterior wall the bladder was well covered by peritoneum, but the anterior wall was extra-peritoneal. Had the sac been opened at what had appeared to be its fundus, the incision would have been into the bladder. The sac was closed by purse-string suture, the stitches following closely the margin of the internal ring,

and as close as considered safe to the junction of the bladder and peritoneum. The stump was then turned towards median line, after freeing the bladder wall from adhesions, and fastened under the internal oblique muscle; the canal was closed by the Bassini method. Recovery prompt, with no recurrence.

Case XVI.—Extra-peritoneal (Card No. 1309), February 5, 1906. M. P. B., age 41; six feet tall, weight 195 pounds (referred by Dr. J. Hoffman, Jersey City). Right inguinal hernia. eight years' duration; truss worn; retained hernia, but painful.

Operation was performed at Murray Hill Sanitarium, Dr. George E. Doty assisting. The canal was filled by fat which covered the sac very thickly at its lower part. The sac was opened near the internal ring and the bladder, uncovered with peritoneum, found dragged over the pubic bone. The sac opened was closed by catgut, the bladder freed and pushed well to the median line, and the canal closed by the Bassini method. Patient left on 14th day perfectly healed. There was no recurrence.

Case XVII.—Extra-peritoneal both sides (Card No. 1340) April 17, 1906. C. R., age 25 years; medium size and rather thin. Double inguinal hernia, for several years, size of hen's egg on either side. Trusses worn, but always with discomfort.

Operation was performed at the Post-Graduate Hospital, Dr. Runyon and staff assisting. On opening the right side the hernial sac, found in the canal, was continuous with a larger tumor at its lower part. The upper sac was opened and the lower mass diagnosticated as an extra-peritoneal bladder hernia. The upper sac opening was closed, the bladder was freed of adhesions and carried towards median line, and the canal closed by the Bassini method. An exactly similar condition existed on the left side and was treated in the same way. Both closures were difficult, but apparently effective. The patient left the hospital on the 14th day perfectly healed. There was no recurrence to January, 1908.

Case XVIII.—Extra-peritoneal (Card No. 1353), May 19, 1906. S. H., age 53 (referred by Dr. Honeyford, Hudson, N. Y.). Right inguinal hernia, five years; size English walnut; elastic truss worn but hernia not retained.

Operation was performed at the Post-Graduate Hospital, Dr. Smith and staff assisting. Some fat and a sac bulging in the upper part of canal was found with a tumor low down on the inside of the cord. The upper sac was opened and the lower protrusion found

to be extra-peritoneal protrusion of the bladder. The sac opening was closed by purse-string suture and the bladder fastened well towards the center by suture through the fatty covering. The canal was closed by the Bassini method. The patient left the hospital on the 14th day perfectly healed. There was no recurrence one year later.

Case XIX.—Extra-peritoneal (Card No. 1401), December 13, 1906. M. W., age 38 (referred by Dr. Abrams). Double inguinal hernia; previous history not known.

Operation was performed at Post-Graduate Hospital, Dr. Smith and staff assisting. On the right side a true direct sac was found, but the lower, and larger, part of the tumor was the bladder uncovered by peritoneum. The sac opening was closed by purse-string suture; the bladder was freed from adhesions and stitched by fatty coverings to the under surface of the internal oblique well towards the center of the abdomen, and the canal closed by the Bassini method. The patient left the hospital on the 14th day perfectly healed.

Case XX.—Extra-peritoneal (Card No. 1442), March 16, 1907. A. J. C., age 45 (referred by Dr. S. G. Gant). Right complete inguinal hernia of supposed recent origin.

Operation was performed at Murray Hill Sanitarium, Dr. George E. Doty assisting. A small oblique inguinal sac and some fat was found in upper part of the canal. Just above the pubic bone and inside of the cord was protruding a small, fatty tumor the size of a small English walnut. The anterior surface could be easily traced down to the bladder wall. The upper sac was removed, the bladder freed and pushed towards center of abdomen.

The patient had some irritation of the bladder previous to operation, and on the following day developed a sharp cystitis, which lasted for nearly a week. As there was very little handling of the bladder diverticulum, it is not known why the cystitis should have developed.

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THE USE OF UTERINE DILATATION.

BY BACHE MC E. EMMET, M.D.
Professor of Diseases of Women, New York Post-Graduate Medical School and Hospital.

For a long time past it has struck me that the method of examination of the interior of the uterus by the finger, preceded by full forcible dilatation of the cervix, is not sufficiently insisted upon in our teaching of gynecology; nor is it practiced as much as I believe it should be, to establish a diagnosis in all cases of enlargement of the uterus or suspected internal disease, before resorting to operation; this latter often being ill-chosen because the diagnosis is not positive or is faulty. Further, I wish to make the point that this method may serve for several purposes of treatment, once the nature of the case is fully determined by digital exploration as above indicated.

In the earlier days of gynecology, such information as may be gained by the use of the finger within the body of the uterus was much more sought than seems to be the fashion of the present time; yet, such practice was attended by difficulties far greater than we now have to contend with, inasmuch as the means of dilatation were less perfect and more harmful. The strong dilators had not yet made their appearance, so that we were compelled to attain our end by the use of sponge tents, or tupelo; the one positively foul and dangerous, the second equally tedious and painful. Further, the use of either of these required at least twenty-four hours to open the canal sufficiently to admit the fore-finger. This entailed the danger of blood-poisoning, in spite of the use of antiseptics, and produced a softening of the tissues conducive to absorption and unfitting them to bear the strain of further dilatation which might be required by a finger larger than the tents, or of instruments which might be used within the body of the uterus.

The development of the steel dilators, or, possibly, should I say, their general use, made it evident that they possessed advantages over other means hitherto at hand, even of the gradual packing with sterile gauze; but these advantages seem to have been limited to time and cleanliness and not extended to that of space; its utility was, therefore, largely confined to

purposes of curetting. Now, it is true that no text-book has failed incidentally to recommend dilatation for the purpose of digital examination, but I hold that the precept has not been emphasized nor sufficiently dwelt upon to induce every practitioner to value it as a ready and safe means of acquiring full information of conditions on the inside of the uterus.

Of course, I have no idea of making of this measure a trifling proceedure which should be undertaken lightly or without due regard to its importance, involving, necessarily, every antiseptic precaution as regards patient, instruments and self. Profound anesthesia is a necessity, as well as a skilled assistant. In fact, we may, from the first, assume that extreme dilatation and exploration by the finger is but the beginning of, possibly, a larger operation which we mean to carry out at the same sitting, and we prepare the patient and ourselves accordingly; whether it be for a mere curetting or securing of a specimen for the microscope, or removal of growth of whatever character, from a polypus to a fibrous tumor, or the uterus itself.

I shall mention, in passing, some points in this connection which are not generally alluded to, but which may well be incorporated here. They are, first: The operator should recognize when he is dilating a cervix that the character of the tissue varies much; dependent upon the age of a patient, whether or not she has borne a child, whether there have been injuries of parturition or long continued disease of the cervix. He should proceed slowly, giving abundance of time for very firm tissue to yield gradually, without tearing, and having a care lest tissue which yields very readily, tear also. Extensive injuries reaching beyond the uterus itself have occurred, so that the operation of exploration has had to be abandoned for one of emergency, for repairs. Secondly, the operator, when about to use the curette, should recognize that the uterus relaxes markedly when a patient is deeply anesthetized. I believe that obstetricians are thoroughly cognizant of this point, and the careful one attending a confinement will not push his chloroform to the point of arresting uterine contractions. Furthermore, one should appreciate that a uterus which is being curetted relaxes still more, as if paralyzed. I have always attributed this further loss of tone of the muscle to the traumatism produced by the curette and, probably, the two causes have a share in producing it.

This point should be heeded, because the possibility of piercing the uterine wall is always present. This injury occurs sufficiently often to create alarm even when a curette is used for minor purposes. The organ is readily perforated even in expert hands, and, considering that a uterus becomes so relaxed that its wall seems to give, before apparently reasonable pressure of the instrument, I always urge caution upon the beginner and instruct him either to follow the curette by carrying a finger up alongside of the uterus, per vaginam, in order to recognize its position, or to grasp the fundus from the outside and so estimate just where he is working,

After making a thorough exploration of the interior of the uterus, one appreciates also outlying conditions which may call for present or subsequent attention. One is then prepared to determine upon a line of action.

If the dilatation has been *well* done, it matters little that it proves sufficient to admit a large finger quite freely through the inner os, as no possible harm can follow; indeed, I think it better so than to bore a way through a tight place. Now one may do little, or much, dependent upon the case.

I am so confident that, failing to utilize this method, major surgery has frequently been invoked by very competent men, when minor surgery would have sufficed, that I never fail to enjoin upon my students when uncertain of the character of the inside " carry the tip of your finger to the fundus of the uterine cavity."

I have known of hysterectomy being performed in case of uncontrollable hemorrhage, when a post-operative inspection revealed a small mass at the fundus, no more than could have been very readily removed by a curette. I have witnessed a moderate dilatation of the cervical canal, followed by a vigorous curetting which removed granulations of good size, and improved the local condition in so far that persistent bleeding and an abundant mucous discharge was abated (glandular endometritis), but, within three weeks a fibrous polypus, the size of a plum, presented at the external " os " demanding removal. It would have been more gratifying to all parties had the operator dilated fully to pass the finger and recognize the true state of things before undertaking to curette.

Dilatation to an extreme degree offers us so great a facility

for operating within the uterine body, that I greatly wonder it is not more resorted to. Apart from polypi, moles, hydatids or other products of conception, fibrous growths, submucous and even mural, may readily be grasped, if one has explored the cavity with the finger and can, therefore, easily place the tenaculum or the forceps upon them with accuracy.

In the case of small fibroids half buried or just presenting above the surface, one may be enabled to gradually work them out of their bed by the aid of an assistant holding the fundus in his hand and pressing intelligently with one or two fingers upon indurated spots or the designated growth from within. As traction and twisting is practiced, the assistant will recognize a tendency on the part of the uterus to invert at such a point, but he may still crowd the growth toward the uterine cavity and make it project, so that, as it advances, uterine tissue will contract back of it and fill its bed.

I am partial to the sponge-holder for the purpose of such removal. The entire blade is a fenestrum and secures for the operator such a hold that he may twist the growth upon its axis until the mucous membrane covering is formed into a pedicle which soon yields, and the growth is extracted, provided the canal be sufficiently open. Otherwise, one may have to cut up the tumor and extract it piecemeal or split up the cervix.

This procedure may be repeated, subject to the judgment of the operator, until all such as can be crowded down are removed. I think that this minor surgery may find a valuable place in our work, and save many a woman from taking extra risks, until such time as she may be compelled to, on account of subserous growths.

Another purpose for which I would advocate the systematic use of the uterine dilator is the treatment of so-called chronic metritis (subinvolution) to be used frequently, under aseptic conditions, of course; the effect sought being the establishment of uterine contractions to follow the dilatation, thereby securing a tone of muscle and blood vessels which will diminish weight, lessen the menstrual flow, relieve pain and allow of restoring the uterus to its proper position in the pelvis. This method is the same as we sought in old times to bring about by the repeated use of sponge tents; and is quite similar to that which masseurs adopt in developing other muscles, viz.: opposing one force to another.

It is a good adjunct to other methods which still prevail, and, I believe, if practised to a moderate degree every three or four days, for a short while, it may prove a quicker means and fairly displace them.

18 East 30th St.

A REMINDER THAT FATAL OR SERIOUS RESULTS MAY FOLLOW OPERATIVE TREATMENT OF BUBOES.

BY EUGENE FULLER, M.D.
Professor of Venereal and Genito-Urinary Surgery, New York Post-Graduate Medical School and Hospital, etc.

As buboes are commonly supposed to represent a condition or, perhaps, better, a complication devoid of any special danger to life, and as the surgical operative treatment of such of them as may require it is classed under the head of minor surgery, it is, perhaps, natural that little or no emphasis has been laid on the dangers which may, and, I am inclined to think, do without great infrequency result in connection with this branch of surgery. When, for instance, death follows what is supposed to be any minor operation, the surgeon in the case is naturally chagrined and little apt to publish the fact. Having given much thought to the subject, it seems to me that death or serious results following such operations can be greatly reduced, if not practically eliminated. In order that this should be accomplished, it is necessary to raise the surgery in this connection from its low minor level to a more important and higher one. As a first step in this procedure, the student should refresh himself as to the regional anatomy and be made familiar with the fatal or serious results which may follow the surgery of this part. Being thus acquainted with possible dangers, the steps to avoid them will then be appreciated and should be considered.

Buboes generally lie just above Poupart's ligament; less commonly below it and sometimes over it. In cases of active suppuration, a fairly extensive area both above and below is frequently involved. The deep limits of such suppurations are determined by the external oblique fascia, which constitutes the outer layer of the abdominal fascia, Poupart's ligament and the deep fascia covering Scarpa's triangle. Over these structures Nature, if left to herself, throws out a barrier of cellular infiltration and granulations which generally is effective in protecting the important parts lying beneath these fasciæ from invasion by the suppurative process. Such suppurations, thus checked from extending in a deep direction, tend to burrow subcutaneously and to point externally. Just below the abdominal fascia

is the important lymph space, limited externally by the abdominal fascia and the fascia propria of the pelvis and internally by the peritoneum. In this lymph space, just under the abdominal fascia, lie the external iliac artery and vein, while between the layers of the fasciæ and still more exposed are the epigastric and circumflex iliac artery and vein—branches of the external iliac vessels. The femoral artery and vein lie in Scarpa's triangle just under the fascia and, consequently, much nearer to the surface than the external iliac vessels.

The dangers in connection with the surgery of buboes lie in two directions. First, in a direct or indirect injury to the blood vessels mentioned, and second, in an inflammatory invasion of the lymph space between the fascia propria of the pelvis and the peritoneum, the infection having entered through the abdominal fascia. Direct injury to blood vessels lies in an actual wounding, during operation, of the femoral artery or vein, of the external iliac artery or vein, or of the epigastric or circumflex iliac artery or vein. The accidental opening of one of these major vessels during an operation of this nature is, I am probably safe in saying, a fatal happening. A similar partial injury to the wall of one of them, there being at operation no actual opening, is generally fatal through secondary hemorrhage. A cutting into the branch vessels mentioned is, of course, not nearly so serious, as ligatures can be applied; still, the danger from secondary hemorrhage is considerable.

The danger from indirect injury to these blood vessels lies in unduly exposing their walls during operation through removal of the connective tissue and the fascial coverings, thus allowing the infective process causing the bubo to come in direct contact with them. Infection coming in such direct contact with these blood vessels may cause septic phlebitis or, through ulcerative action, secondary hemorrhage.

Inflammatory invasion of the deep lymph space already mentioned is generally occasioned by two surgical errors. First, at the time of operation the basement structures of the bubo are so removed by curettage or by cutting with curved scissors that the abdominal fascia is laid bare and, probably, also actually damaged. The second error lies in making a too narrow cutaneous opening. The combined result of these surgical faults is that the cutaneous opening closes prematurely to a narrow

sinus, the original bubo cavity being left a pus-pocket, the contents of which finally burrows through the injured abdominal fascia and enters the lymph space.

The cases of immediate death in connection with bubo surgery wherein the knife of the operator, during the attempt to open the abscess by a single quick incision, penetrates too deeply and lays open a main trunk blood vessel are, of course, infrequent. In such cases, as a rule, the incision has been attempted without an anesthetic and the patient on the first plunge of the knife point has sprung upward from the table or has struck or seized the surgeon's arm. Such actions on the part of the patient may, of course, cause any surgeon to lose control of his knife and so be unaccountable for the faultiness of the incision. Still, the fact that many individuals cannot control bodily actions in the event of sudden and severe pain should always be in a surgeon's mind before an attempt is made to open a bubo in this manner; and not only should the arms be held or so guarded that the operator cannot be seized, but the knife-point should be directed superficially or laterally, rather than downward, while the end of the little finger of the operating hand should rest, before and during the performance of the incision, on the body of the patient.

The cases which I will now record in short detail, serve to illustrate the preceding remarks.

Case I.—A strong, vigorous young man of about 25. There was in this case a history of chancroid, followed by a very virulent bubo, which had been freely opened and thoroughly curetted, especially at its base. The cavity had been then quite tightly packed with antiseptic gauze. The case progressed uneventfully and apparently favorably until the sixth day after operation, when, all of a sudden, a very excessive hemorrhage occurred. General pressure was promptly applied, and the surgeon quickly responded. Pressure on the main arterial trunk above the wound was made and the dressings removed. The bleeding, arterial in character, was found to come from the floor of the cavity, bursting through the newly forming granulations. After several attempts, an arterial clamp was so fastened as to control the hemorrhage. It was impossible, however, to pass a ligature below the clamp. It was evident also that the clamp had a hold on treacherous ground. The hemorrhage in this instance

seemed to come directly from an ulcerative perforation of the main artery. The clamp did not block the artery, thus shutting off blood supply to the leg, but so constricted the little connective tissue remaining about the vessel as to stop the bleeding. Eight hours after the first secondary hemorrhage, another occurred which proved fatal. This final hemorrhage was from the same site as the first, the clamp failing to maintain its hold. When my attention was called to the case shortly after the clamp had been applied to control the first secondary hemorrhage, I felt that the prognosis was necessarily fatal, since to guard against a recurrence of hemorrhage more effectually by ligation of the arterial trunk would not only invite gangrene of the leg and death from general sepsis, but even such treatment would not guard against a further hemorrhage from extension of the infectious ulcerating process to the arterial wall above the site of the ligature.

Case II.—A young man of good physique, 22 years of age. He, too, had a virulent bubo secondary to chancroid. This case had been subjected to operation, just as had Case No. I; only in this instance, besides a curettage of the base of the bubo, curved scissors had been employed to trim off shreds of connective tissue. The bubo cavity had then been tightly packed with gauze. After this operation the patient had been very comfortable, and for three days had shown practically a normal temperature. On the 4th day the temperature began to rise and deep tenderness and swelling in Scarpa's triangle developed, quickly followed by phlebitis and swelling of the leg. On the 5th day secondary arterial hemorrhage occurred, which was controlled, as in Case I, by the application of a clamp. Gangrene of the leg and thigh began, and the patient died the following day of acute sepsis, hastened by the debility caused by the hemorrhage.

Case III.—A young woman, 20 years of age. She was admitted to the City Hospital in a very low condition from general sepsis. In the right inguinal region there was a sinus, the only remaining surface indication of a preëxisting bubo. She gave a history of a bubo in that position which had been operated on over two months previously. Just how extensive the operation had been, could not be learned from the patient. The scar, however, showed that only a very narrow external opening

had been made and, from the fact that the cutaneous structures were directly adherent to the abdominal fascia, made it most probable that all the structures of the base of the bubo down to the abdominal fascia had been removed. In the right loin and perinephritic region, there was great tenderness, resistance to pressure and some tumefaction. A long silver probe, introduced into the surface sinus marking the bubo operation, found its way through an opening in the abdominal fascia directly into the deep lymph space between the fascia propria of the pelvis and the peritoneum. An incision into the right loin was then made, which gave vent to a very large collection of pus and opened into an extensive extraperitoneal cavity. In the lower pelvic portion of this cavity, the end of the probe projected which had been passed in at the sinus left by the bubo operation. The upper limits of the cavity burrowed about the right kidney, exposing the liver and possibly even mounting to the diaphragm. This immense cavity was thoroughly drained, one of the tubes passing along the original path of the burrowing, through the perforation in the abdominal fascia, along the deep pelvic lymph space and out at the loin incision. The patient, however, at the time of my operation, was in too low a condition from general sepsis to rally, death occurring some few hours later.

Case IV.—A man, 26 years of age, entered my clinic at the Post-Graduate Hospital. He stated that a few days previously he had been discharged from an institution wherein he had undergone an operation for bubo. He was weak and feverish, and complained of indefinite deep pain in the left pelvic region and of a stiffness of the left thigh. The bubo had been on the left side. Two months previous to the occurrence of the bubo he had contracted syphilis, and shortly after that gonorrhea. He had worked hard and, as he thought, had bruised himself in the left inguinal region shortly before the swelling there became so acute as to demand operation. On examination I found a small external sinus connecting with a large pus pocket which occupied the site of the previous bubo. On making an incision, enlarging freely the track of the sinus, the pus pocket was fully exposed. It was then evident that all the lymph glands and the surrounding connective tissue, right down to the abdominal fascia, had been removed at the time of operation through an external opening so narrow that a premature surface closure had taken place.

From the constitutional symptoms existing, I was fearful lest a deep burrowing through the abdominal facsia and into the underlying lymph space had occurred and, accordingly, with a probe I gently prodded the thin granulations which were forming to protect the surface of the fascia. My suspicions were verified, the probe end finding a narrow opening in the fascia through which the shaft of the instrument dropped four inches without any manipulation. Along side the shaft of the probe, pus welled up from the deep lymph space. The patient was admitted to my hospital service. Under an anesthetic it was found that the burrowing, after reaching the posterior lateral floor of the pelvis, had turned upward toward the loin, as in Case III. A left loin incision was consequently made just above the posterior limit of the crest of the ilium. The finger introduced into this incision and turned downward over the crest of the ilium into the pelvic cavity, could detect the movement of the end of the probe introduced into the lymph space through the abdominal sinus. This showed that, as yet, the upward limit of the burrowing was still below the iliac crest. Still, by a little pressure, the fingertip was made to meet the end of the probe, and a drainage-tube was threaded over the probe and passed in at the sinus opening through the abdomial fascia down into the deep lymph space between the peritoneum and the fascia propria of the pelvis and out at the loin. The patient remained in the Post-Graduate Hospital nearly two months. His fever soon disappeared after the drainage described was established. He gained in flesh and, after the discharge along the tube had apparently become almost *nil*, the tube was removed and he was discharged from the hospital. Several months afterwards the patient entered my service at the City Hospital in a very low septic condition. He reported that for six weeks after leaving the Post-Graduate Hospital, his condition had been satisfactory, after which the fever began again, together with the pains and discomfort in the left pelvic region. Then a new symptom developed in the form of swelling and tenderness of the left buttock and hip. This swelling had rapidly increased and had extended downward, involving the thigh to the knee and upward as far as the lower ribs. When the patient entered the City Hospital, his condition, due to general sepsis, was so low that I had little hope of his surviving an operation, the object of which was to radically drain the interior of the pelvis. The patient con-

sented to a further operation, and chloroform was administered, after which it was possible thoroughly to examine the condition. In the first place, the original incision in the left loin above the posterior edge of the crest of the ilium was reopened and the drainage-tube replaced, which entered the extraperitoneal pelvic cavity from this opening and extended upward, passing through the abdominal fascia along the track of the original sinus. As this tube did not seem to thoroughly drain the dependant portion of the pelvic cavity, the patient was next put in the lithotomy position and a free lateral incision made to the left of the rectum, opening the ischiorectal space. The ischiorectal fat was incised, laying bare the levator ani muscle. A probe was then passed through the sinus-opening in the abdominal fascia and into the bottom of the deep pelvic lymph space, where its end was made to maintain firm pressure against the pelvic fascia lying above the left levator ani muscle. A cut was next made from the ischiorectal incision through the levator ani muscle and the deep pelvic fascia on to the end of the probe. This being done, the probe end was passed along and out at the ischiorectal opening. A drainage-tube was then passed along the ischiorectal incision and into the extraperitoneal pelvic cavity. Long forceps were next introduced at the opening in the loin above the crest of the ilium, and the end of the tube introduced at the ischiorectal opening was seized, drawn along the pelvic cavity and out at the loin. This low level second tube certainly gave the freest drainage to the deep pelvic space and allowed irrigation from the loin out by the rectum, the lowest possible level. Attention was next turned to the tumefaction in the left gluteal region, and an incision was made about four inches long, beginning above, half way between the posterior superior spine of the ilium and the great trochanter, from which point it extended downward over the course of the sciatic nerve between the tuber ischii and the great trochanter. This incision, after piercing the deep gluteal fascia, opened up a very foul abscess cavity which had burrowed downward and encircled the capsule of the hip-joint. On following up this cavity with the finger, it was found that it communicated with the deep pelvis through the great sacrosciatic foramen, and the finger passed into the pelvic cavity through this foramen showed also extensive necrosis of the pelvic bone about the great sacrosciatic notch. Two large drainage-tubes were then passed in at the gluteal opening, through the

sacro-sciatic foramen and into the pelvic cavity, and were there maintained. By this operation it is seen that the pelvic space was drained in four independent directions. 1st, through the opening in the abdominal fascia; 2nd, through the opening in the loin; 3rd, through the opening in the ischiorectal region, and 4th, through the gluteal opening by way of the sacrosciatic foramen. This represents the most perfect and complete drainage possible of the deep pelvic space. I have not been able to find in medical literature, the records of a case similar to this one. I have no doubt, however, that many such instances of suppurative burrowings have occurred and have gone by default, as it were, no such radical surgery having been attempted.

The patient recovered from the operation, gradually throwing off his septic condition. The existence of the necrosis in connection with the pelvic bone made it very evident that, at best, his recovery would be most tedious. Now, at the end of two years since the preceding operation, the patient is still at the City Hospital going about aided by a crutch, due to stiffness and pain about the left hip. A discharge, owing to the bone necrosis, still persists from a sinus marking the site of the gluteal incision, and also from the original one through the abdominal fascia.

After the preceding recital, no one can fail to see the importance of the following rules in connection with bubo surgery. In making a primary incision be careful not to enter the knife-point deeply. Make the external opening so free that there can be no premature closure of the external wound. In order to assure this, a cruciform external incision is often wise or necessary. In the case of a virulent bubo, never curette or disturb the necrotic contents, but allow a natural demarcation to take place between the healthy peripheral tissue and that sloughing as the result of the bubonic infection. In the case of suppurative tubercular buboes, after exposing them by free incision, enucleate the tumefied glands by means of some blunt instrument, the back action of the operator's thumb often being the most serviceable agent. After so removing the glands, do not cut away with curved scissors or with a curette the sloughing, shreddy tissues left at the base, but leave them for a natural detachment. The cavities of these last buboes can be loosely packed with iodoform gauze.

RUPTURE OF THE UTERUS THROUGH THE CÆSAREAN CICATRIX.

BY GEORGE L. BRODHEAD, M.D.
Professor of Obstetrics, New York Post-Graduate Medical School and Hospital.

In November, 1906, the writer had the misfortune to lose a patient by rupture of the uterus through the scar of a Cæsarean section which he had performed in December, 1904. With the object in view of ascertaining the frequency of this accident, the mortality, etc., the literature has been searched, cases have been recorded and deductions of value have been drawn.

Frequency.—It is wonderful to note the great difference in the frequency of rupture after the old Cæsarean section, as compared with the results following the new or modern operation.

Krukenberg,[1] in 1886, stated that, after the old operation, 50 per cent. of all cases resulted in rupture of the uterus during subsequent pregnancies. He reported 20 cases of rupture through the scar, with a mortality of 50 per cent. On the other hand, Olshausen, in speaking of the Prüsmann-Henkel case (No. 12)[19], stated that it was the only one of scar rupture in at least 120 Cæsarean sections. In the literature of the last five years we have found the records of 18 cases, to which we have added two hitherto unreported cases, making a total of 20. Among these will be found one case (No. 11)[18] where the uterus was not sutured at the time section was performed, and, properly, such a case might be excluded from our statistics. When one considers the large number of sections which have been performed, many of which have not been reported, the accident is seen to be of comparatively rare occurrence. In spite of the infrequency, however, the nature of the accident is so grave that one must keep the possibility of the emergency constantly in mind.

Etiology.—Much has been written in regard to the cause of rupture, but, briefly, there seem to be two factors in the etiology. One is the natural weakness of a cicatrix, whether in the uterus, abdominal wall or perineum, and the other is the invasion, so to speak, of the musculature of the uterus by foci

of decidual cells. In Prüsmann's case, decidual tissue had invaded the muscular tissue about the incision and had even extended to the serosa. Sinclair[2] believes that if silk were used for suture of the uterine wound, rupture would seldom or never occur; but in at least two of the reported cases, those of Woyer and Everke, the rupture occurred in spite of silk sutures. We believe that if good approximation of the edges of the wound is secured, and interrupted sutures of chromic catgut are used, the result will be perfect, as far as complete union of the parts is concerned.

Diagnosis of Rupture.—In many cases, especially in the complete variety, there are the typical symptoms of sudden cessation of pain following a severe uterine contraction, with evidences of shock, increase in the pulse rate, vomiting and cold sweat. The fetal parts can be palpated with greater ease, the fetus is more movable, and the contracted uterus may be felt. By vagina, the presenting part has slipped away from the brim, and there may be external hemorrhage. In several of the reported cases, however, the rupture was not suspected until the operation for section was performed, and in one case a diagnosis of appendicitis had been made. Evidently, the condition was not discovered in some cases, for operation was not performed until hours had elapsed since the rupture had taken place. In our opinion, labor after Cæsarean section should be anticipated, but if one is called in to see a patient who is having severe uterine or abdominal pain, at or near term, the abdomen should be opened at once.

Repeated Caesarean Section.—Wallace,[3] in his exhaustive monograph on repeated section, collected 96 cases, 43 of which had had two operations, 15 had had three, one had four, and one, the celebrated Frau Rittgen case, had five, the last resulting fatally from general peritonitis. Kriwski,[7] in 1905, reported 88 cases, 72 of which had two sections, 13 had three, and three patients had four. Many other instances have since that time been reported, among them that of Sinclair,[2] in which four sections have been successfully performed on the same patient. The mortality of repeated section is stated by Wallace[3] to be 6.45 per cent. (4 in 62), or including death from repeated fundal incision, 7.93 per cent. This certainly compares favorably with the mortality of primary Cæsarean section, which in the hands

of all operators, is between five to ten per cent. In the hands of experts, the mortality of both classes of cases is probably less than five per cent.

Mortality of Rupture through the Scar.—Krukenberg,[1] referring to the old operation, quoted a mortality of 50 per cent. Very different has been the death rate in the cases reported and referred to in the literature of the last five years. Of the 20 reported cases, 16 recovered, three died, and the result in one case (No. 4)[11] is unknown. Note must be made of the fact that in case No. 11[18], the uterus was not sutured at the time of the section, and we have included in our cases of recovery the case of Lobenstine, for when the patient was ready to sit up on the tenth day pneumonia set in, which terminated fatally on the 17th day. This death can certainly not be attributed to the operation. The mortality is, therefore, 15 per cent. In each of the three fatal cases the rupture was complete, the child having entered the abdominal cavity, and in one case laparotomy was not performed until two days had elapsed from the onset of labor. In my own case, the patient was dying when the laparotomy was performed, some five hours after pains had commenced, at the end of the eighth month of pregnancy. When one compares the 15 per cent. mortality with the mortality of rupture of the uterus in general, one is astonished that the figures are so much lower. In recent years the mortality has decreased, but the figures given below give some idea of the appalling death rate from rupture. Dorland[4] collected from the literature of the three years from 1901 to 1903, 50 cases with a mortality of 24 per cent. Ivanoff[5] stated that, in the Moscow Maternity, from 1877 to 1901 there had been 124 cases of which 98 died, giving a mortality of 79 per cent. The same author quotes Klien, who gives 56 per cent. recovery by operative methods, and Kolomenkin who gives 53 per cent. recovery by operation (but excluding cases of laparotomy and suture, 64 per cent.). Lobenstine[6] quotes 37 cases with a mortality of 73 per cent., but states that in the last six cases the mortality was only 33 per cent. The fact must be considered that in the case of a patient who has had a Cæsarean section, the treatment of a subsequent pregnancy and labor is frequently referred to experts, which would naturally account for the smaller mortality. The fetal mortality is very high. Of the 16 cases where the result for the child is known, the child lived in

three, giving a mortality of 81 per cent. The child died in every case of complete rupture and when the child was alive the rupture was small.

Variety of Incision and Character of Rupture.—The incision was longitudinal in thirteen cases, fundal in six and probably fundal in the remaining case of the twenty. Of the three fatal cases, two had had the longitudinal incision, one the fundal. In recent years the anterior incision has been used much more often and, indeed, there is no reason to believe that there is any advantage in the fundal incision. We believe that the greater frequency of rupture with the longitudinal incision is due to the very much greater frequency of that incision. The rupture was complete (by which we mean that the ovum passed out of the uterus into the abdominal cavity) in fourteen cases, partial in five and in one case unknown. All of the partial and eleven of the complete cases recovered.

Treatment of Rupture.—Hysterectomy was performed in twelve cases, with two deaths. Suture of the wound was the treatment in six cases, all of which recovered, and in my own case the patient died before any treatment could be instituted. In Schütte's case (No. 9)[16], the operation was done some days after the rupture, and the uterus was well involuted. Whether the uterus was sutured or not we do not know. The results where the rupture was sutured were all favorable, and yet many operators perform hysterectomy for the treatment of the condition. In the hands of experts either plan of treatment will in most cases be successful, but in clean, simple cases where the tear is not ragged, there is no reason why the uterus should not be sutured. Certainly, in the hands of one who is not expert in abdominal surgery and when the patient is in bad condition, the suture offers a quicker and safer method of treatment than hysterectomy. Quoting again from the statistics of Dorland[4], which refer to rupture of the uterus in general, we find that the uterine wound was sutured in ten cases, only one of which died. Five of the nine women who recovered after suture became pregnant again. In three of these, labor was normal, in one case forceps were used, and the fifth was terminated by induction of labor at the thirty-third week. These figures, which indicate a mortality of only 10 per cent. after suture and the successful termination of pregnancy in five cases, certainly indicate that the plan of treatment

by suture is eminently successful and, therefore, should, as a rule, be advocated. According to the statistics of Schultz, Valenta, Klien, Kolomenkin, Dorland and others, the operative treatment of rupture in general offers the best results, and in this opinion, especially where the patient is surrounded by the proper conditions and is under the care of a skillful operator, we must agree. Speaking again of rupture in general, we believe that in the absence of the above conditions, when rupture has occurred, the fetus should be extracted by forceps, version or craniotomy, after which the uterus should be tamponed. If hemorrhage continues, or if the necessary preparations can be made, abdominal section should be performed, and the uterus sutured or removed, as the operator may elect.

Prophylaxis, Including Sterilization, Time of Operation and Technique of Caesarean Section.—The question has arisen: "should a patient, in the presence of absolute indications for Cæsarean section, be sterilized at the time of the first section?" It seems to me that in the absence of any condition, such as carcinoma or fibroids, which in itself would indicate the removal of the uterus, the question of future pregnancy should be answered before the Cæsarean is performed. The danger of repeated section and the possibility of rupture of the uterus during pregnancy and labor, should be put before the patient plainly, and, in our opinion, if the woman knowing the facts of the situation elects to undergo repeated Cæsarean sections, she should be allowed to do so, and, indeed, she may do so without great risk, if we may judge from the figures which have been already stated. If the patient is opposed to the risk of a second or subsequent section, sterilization should be performed. The simplest way of is doing this is to resect a portion of each tube. Ligature alone is not sufficient, as the case of Galabin (No. 6)[13] will prove. Sinclair[2] is violently opposed to sterilization, quoting the small mortality of repeated section and the infrequency of rupture of the uterus. He says: "Sterilization and hysterectomy are rapidly entering the limbo of deplorable episodes in the history of obstetrics and gynecology." Galabin and Green are equally emphatic in their views on the subject.

With reference to the time at which operation should be performed, we believe that it is better to anticipate labor by a week or ten days, in order that the operation may be performed at a

convenient time, with proper surroundings and good assistants. Should labor set in earlier than had been expected, we advise that the Cæsarean operation be performed as soon as possible. In many of the recorded cases labor had been in progress for a number of hours, and each hour of labor adds to the danger of rupture. Experience has shown that results are fully as good when the operation is performed before labor commences, and, indeed, there are many advantages to be gained by operating at an elected time and place. Finally, we refer to the technique of the section, as entering into the prophylaxis of the repeated operation and subsequent rupture.

My method of performing the section is as follows: The usual preparations for laparotomy having been made, the bladder is emptied by catheter, and the cervix, if closed, should be dilated to the extent of two fingers. An assistant then makes pressure at either side of the abdomen, thus bringing the abdominal and uterine walls closely together, and forcing the uterus well forward in the median line. An incision is then made, about six inches in length, in the median line of the abdomen, beginning at a point about three inches above the umbilicus. Sterile pads are then placed at either side of the incision, and then we make a median uterine incision of the same length as the abdominal. The child is seized by one or both feet, and quickly extracted. If in making the incision, the placenta is encountered, the hand is quickly forced through it, the child is extracted and the placenta removed at once. An assistant seizes the uterine arteries, and makes digital pressure, sufficient to shut off the uterine circulation. The umbilical cord is then cut, and the child is handed over to an assistant. While this is being done, an assistant brings the uterus up into the abdominal wound, and protects the peritoneal cavity with gauze pads. The placenta and membranes are removed manually, and the uterus is dried with a gauze pad. If the uterus is well contracted no packing is necessary, but if it is soft and shows a tendency to relax, the cavity should be packed with sterile gauze. Interrupted sutures of No. 2-20 day chromicized catgut, are passed through the inner two-thirds of the muscular coat, down to, but *not including* the mucous membrane. Another layer of sutures of the same material is then passed through the peritoneum and the outer portion of the muscular coat, and finally a continuous suture of No. 1

catgut is taken in the peritoneal coat, in order to close in all raw surfaces, and to prevent contamination from the uterine cavity. All blood and clots are then removed from the peritoneal cavity, which is then filled with hot normal salt solution, after which the abdominal incision is closed, layer by layer, in the usual manner.

Wallace[3], in his complete monograph, stated that (1) complete utero-parietal adhesions render repetition of Cæsarean section simple and easy; (2) that all sections should be performed with a view to ultimate pregnancy; (3) that this can be done by the adoption of means to ensure complete utero-parietal adhesions. On the other hand, the presence of any marked form of adhesions other than complete utero-parietal, tends to render repeated section more formidable. He said also that to Michaelis (who, in 1836, performed the operation for the fourth time on the same patient the last three operations being extraperitoneal), Sinclair and Spencer belong the credit of recognizing the truth of the principles just enunciated. Speaking of adhesions, Sinclair[2], in his recent article on repeated section says: "Accumulating experience points to the conclusion that the patient is safest when, during a second or subsequent operation, the adhesions are most carefully preserved from interference." He then reports a case on whom four sections have been made. After the first section the uterus became adherent to the abdominal wall, as after a ventrofixation. The second and third sections were practically extraperitoneal (the opening into the peritoneum being exceedingly small), and the fourth section was extraperitoneal. Sinclair has had no opportunity during the last five years, of operating a second time in any case where ventrofixation had been intentionally performed at the time of the first section; but he thinks that the evidence from numerous reported cases points to the conclusion that if adhesions occur, and are respected, the danger is greatly diminished. He has seen no reference to a fatal result in such a case. His method of operating is as follows: After the usual section, the uterine wound is closed with silk. Fine silk sutures, one on each side, are introduced low down in the vesicouterine fold, well beyond the bladder laterally so as to close the fold and prevent intrusion of the intestines at some future time. He then passes a fine silk suture through the parietal peritoneum opposite the lower end of the

incision, and then takes hold of a layer of uterus well out from the margin of the uterine wound. The suture is then passed back through the peritoneum and tied. This method of suturing is continued symmetrically on both sides, until the uppermost suture is slightly above the level of the lower margin of the corpus uteri. Then a stronger silk suture is passed through the fascia on one side of the wound, through the peritoneum and across " through a considerable, not very superficial portion of the uterus," then out through peritoneum and fascia and tied as a buried suture. Another suture is passed higher up, so as to include the uterine wound near its upper termination. This secures a wide area of adhesion, and the abdominal wound is then closed. Intestines and omentum are thus excluded from the field of future operations. Sinclair then gives his experience of thirty cases cured of sterility by hysteropexis hypogastria, who have gone through pregnancy and parturition without abnormal symptoms. Further results from this plan of treatment will be awaited with great interest. We have known rupture of the uterus to follow ventrofixation, and time only can decide as to the value of the method.

Conclusions.—1. Rupture of the uterus through the Cæsarean cicatrix is of rare occurrence.

2. With prompt operative methods the mortality is comparatively low.

3. When pregnancy follows Cæsarean section, the patient may be safely delivered again by section in a large percentage of cases.

4. In repeating a section, labor should be anticipated by a week or ten days.

5. If section is to be repeated and labor sets in prior to the time elected for operation, the Cæsarean should be performed as soon as possible after the onset of labor pains.

6. Sterilization may be done at the time of section, if the patient so desires.

7. Suture of the laceration has proven successful, but in some instances hysterectomy will be the method of choice.

Case I., reported by Koblanck.[8] In this patient the anterior incision had been made, and rupture occurred through the scar. The intact ovum was found in the abdomen; the laceration was sutured and the patient recovered.

No. of Case.	Reported by	Date.	Period of Gestation.	Cæsarean Incision.	Variety of Rupture.	Treatment.	Result, Mother.	Result, Child.
1	Koblanck	1895	Full term? labor?	Longitudinal.	Complete.	Suture.	Recovery.	?
2	Woyer.	1896	"Beginning of labor." Term?	"	"	Hysterectomy.	Death.	Twins, both dead.
3	Guillaume.	1896	" In the eighth month."	"	"	"	Recovery.	Dead.
4	Targett.	1900	" Full term? labor?	"	"	"	?	?
5	Everke	1901	" End of pregnancy."	"	?	"	Recovery.	Dead.
6	Galabin.	1902	" Near term."	"	Complete.	"	"	"
7	L. Meyer.	1903	At term in labor.	Fundal.	Partial.	Suture.	"	?
8	Ekstein.	1904	Full term in labor.	"	Complete.	Hysterectomy.	Death.	Dead.
9	Schütte.	1904	At the eighth month.	Probably fundal.	"	Laparotomy, suture?	Recovery.	"
10	Kerr	1904	In last month, in labor	Fundal.	"	Hysterectomy.	"	"
11	Ribemont-Dessaignes and Rudaux	1904	at term in labor (hours)	Longitudinal. (uterus not sutured)	Partial.	"	"	Alive.
12	Prüsmann Henkel.	1905	Full term? labor?	Longitudinal.	Complete.	Suture.	"	?
13	Schink	1905	At term in labor	Fundal.	"	"	"	Dead.
14	Werth	1905	Full term? in labor?	Longitudinal.	"	Hysterectomy.	"	"
15	Wyder.	1905	At term, in labor 20 hrs.	Fundal.	Partial.	"	"	"
16	Paddock	1906	At term, in labor 24 hrs.	Longitudinal.	Complete.	"	"	"
17	Mabbott.	1906	At term, in labor 20 hrs.	Fundal.	Partial.	Suture.	"	Alive.
18	Schneider.	1907	At term, in labor 7 hrs.	Longitudinal.	"	"	"	"
19	Lobenstine.	1908	At term, in labor 18 hrs.	"	Complete.	Hysterectomy.	Recovered from operation but died of pneumonia on 17th day.	Dead.
20	Brodhead.	1908	36 weeks, in labor 5 hours?	"	"	Laparotomy, death of patient following a few minutes later.	Death.	"

Case II, reported by Woyer.[9] Cæsarean section had been performed by Chrobak in 1893, the anterior incision being made and silk used for suturing. Rupture occurred at the beginning of labor. The abdomen was opened at once and twins were extracted from the abdominal cavity. The patient died the same day. At the time of operation it was found that the wound of the Cæsarean section had failed to unite through the entire thickness of the wall. Parts of the muscular wall had never united.

Case III, reported by Guillaume.[10] This case is of unusual interest because of the fact that rupture occurred in the eighth month of pregnancy. Cæsarean section had been performed three years before, the anterior incision had been made and the wound had been sutured in layers. When the patient became pregnant again, she was advised to have labor induced in the eighth month and the date decided upon was May 25th. On May 20th, at 9 A.M. the patient was seized with severe abdominal pain, especially on the right side. At 3 P.M. the pain was most severe. At 5.30 P.M. the pulse was small and thready and there were vomiting and chills. The abdominal pain was at this time not so intense, and the fetal parts could be distinctly felt, as the child had escaped into the abdomen. Laparotomy was performed a few hours later, and the ovum found intact in the peritoneal cavity. The rupture was found to have taken place in the old scar. The uterus was removed and the patient recovered. Guillaume states that similar cases have been seen by Saint Moulin and Kufferath.

Case IV, reported by Targett.[11] In the Cæsarean operation performed two and a half years before, the anterior incision had been made. Complete rupture occurred and hysterectomy was performed. It is not stated whether the patient recovered or not.

Case V, reported by Everke.[12] In this patient the anterior uterine incision had been made and the uterus sutured with silk. At the end of the succeeding pregnancy there was severe abdominal pain, and the patient was brought to the hospital in collapse. A diagnosis of appendicitis was made, and the abdomen when opened was found to be full of fresh blood. Rupture had taken place in the Cæsarean scar; the uterus was removed and the patient recovered.

Case VI, reported by Galabin.[13] In the Cæsarean operation the anterior incision had been made, and attempt had been made, to sterilize the patient by ligating the tubes. In spite of this the patient became pregnant again, and the uterus ruptured at about full term along the line of the Caesarean cicatrix. The child and part of the placenta had escaped into the abdominal cavity. Hysterectomy was performed and the patient recovered.

Case VII, reported by L. Meyer.[14] Two and a half years after Caesarean section for contracted pelvic, rupture of the uterus occurred at term. Laparotomy was performed and there was found a rupture of the former scar, from which the placenta projected, and there had been considerable bleeding. The uterine tissue was very friable. Apparently, rupture had not been diagnosticated, laparotomy having been intended for simple Caesarean section. The operator was certain that the rupture had

preceded the operation, the accident having occurred probably during severe pains while the patient was in the bath-tub. The fetus was extracted through the rupture wound and the latter was sutured. Recovery was uneventful. In the opinion of the operator, the original Caesarean wound had not healed thoroughly.

Case VIII, reported by Ekstein.[15] Age 33; IV-para first seen June 8, 1904. First two labors ended in perforation, the third in Caesarean section. The patient was warned against future pregnancies (rachitic flat pelvis), but the advice was unheeded. At term pains set in and were extremely violent. After a time fetal movements ceased and the patient was removed to a hospital. There were present meteorism, vomiting, hiccough, small pulse, etc., and the fetal heart sounds were inaudible. Sensitiveness to palpation seems to have interfered with diagnosis, as laparotomy was not performed for two days. Incision was made along the old scar, exposing fetus, membranes and placenta. The uterus was found contracted and ruptured exactly in the scar of the old fundal incision. The dead fetus, placenta and blood were removed and a Porro operation performed. Death occurred one and a half hours after operation.

Case IX, reported by Schütte.[16] Upon this patient Caesarean section had been performed sixteen months before the accident, leaving a fistulous tract from the uterus to the abdominal wall. At the eighth month of the pregnancy which followed the section, the patient fell out of bed, and three days later pain, vomiting, etc. set in, but subsided shortly to reappear in two weeks with evidences of uterine rupture. Laparotomy revealed a macerated fetus among intestinal loops. The uterus was contracted to the size of a fist. The laceration at the fundus was only 3 to 4 cm. long (several intestinal fistulæ formed afterward and were eventually healed by operation.)

The laparotomy wound was not closed outright, but with drainage. The uterus was strongly adherent to the intestines. No mention of closure of the small laceration in the uterus is made.

Case X, reported by Kerr.[17] The patient, a IV-para, had undergone Caesarean section two years before for flat pelvis. Labor had been allowed to proceed until the cervix was fully dilated; then the fundal incision of Fritsch was made, and an 8 lb. living child extracted. Recovery was perfect. On admission to the hospital for a second Caesarean operation, in the thirty-seventh week of pregnancy, the patient stated that she had felt no discomfort since the beginning of pregnancy. She was well nourished and in good condition. After an enema, there was abdominal discomfort, slight pain in the epigastrium, extending upward to the right. The patient fell asleep and slept four and a half hours. Then a bloody vaginal discharge appeared, and the patient complained of slightpain in the right iliac region. Nine hours later the temperature was 97.6° F. the pulse 80, and there was pain all over the abdomen, but it was not very severe; there was no nausea or vomiting. Nine hours later there was considerable abdominal tenderness, but the pusle was good. One hour later the temperature was subnormal, respiration more rapid and tenderness more marked The patient lay with legs drawn up, and there was exquisite

tenderness over the entire abdomen, more marked to the right and below the navel. The fetal parts could be readily palpated, and the presenting part could not be felt through the cervix. The abdomen was opened along the line of the previous scar; a large amount of dark clots was found and the intact fetal sac was lying free in the abdominal cavity, the contracted uterus lying behind and below. The full term fetus was dead. The uterus showed a transverse rupture through the cicatrix of the previous section. The uterus was removed and the patient recovered The interesting point was that there was slight alteration of pulse tension and rate and no collapse, although complete rupture had occurred.

Case XI. reported by Ribemont-Dessaignes and Rudaux.[18] The patient, age 29, had had a difficult labor in 1899, which was terminated by Cesarean section. The woman's condition was so serious that the uterus was not sutured. The peritoneum was sutured with catgut, and the patient recovered. In January, 1904, the woman went into labor again at term. After labor had been in progress for some hours, the patient suffered from very severe pain, and the pulse was accelerated. The fetal parts could now be easily palpated, and the diagnosis of rupture of the uterus was made. Laparotomy was performed several hours later. The uterus was found to be ruptured in the scar of the former section, and a living child was extracted through the rent. The Porro operation was performed, and the patient recovered.

Case XII, reported by Prusmann-Henkel.[19] Age 40; III-para; seen Jan. 21, 1905. First Caesarean in 1894, a second in 1902. Admitted to the clinic shortly before term with symptoms of complete rupture of the uterus. Laparotomy was performed, and the fetus, membranes and placenta were found outside of the uterus, which was moderately contracted. The rupture was found in the old scar, which was sagittal over the fundus and anterior wall. The edges of the rupture were freshened and ten muscular sutures were placed. This case was also complicated with a history of hernia of the abdominal scar, which had been operated on in 1904. The scar of this wound was excised for a finger's breadth. Recovery was smooth.

Commenting on this case, Olshausen states that he has seen but one such rupture in at least 120 Caesarean sections.

Case XIII, reported by Schink.[20] Age 28 years. First seen, July 8, 1902. On Feb. 16, 1899, the patient had undergone Caesarean section for contracted pelvis. The fundal incision was closed by sutures in two planes: (1) a deep suture embracing the peritoneum and musculature; (2) superficial, peritoneum only. The woman became pregnant one year later. At term labor set in, but the child, found to be lifeless, was perforated and extracted. One year later another (the present) pregnancy for which she was admitted at term at the clinic. Strong pains were followed by collapse, necessitating removal to the hospital. There were marked anemia, cyanosis, chills, pulse small, 156–160. Rupture of the uterus was evident on palpation. Laparotomy, July 8, 1902. Incision in the region of the original abdominal scar. The ovum was found intact in the abdominal cavity, and, together with a large amount

of blood, was removed. The uterus was found to have been ruptured exactly in the site of the old fundal scar; at this point the uterine wall consisted wholly of serosa and a narrow strip of muscle. The edges of the wound were freshened in the entire thickness of the musculature, and catgut was used for the mucosa and adjacent musculature. Over these silk sutures were placed, grasping the entire musculature and serosa. The fetus was dead, but the mother made a good recovery, and has not been pregnant since.

Case XIV, reported by Werth.[21] This patient had a pelvis which was rachitic to a high degree. The first labor resulted in a dead child. The second pregnancy (1893) was terminated by Caesarean section with good recovery, the child surviving. The longitudinal incision was made a little to the left of the median line. The third gravidity ended in abortion at the fifth month. The fourth, in 1905, ended in sudden rupture soon after admission to the clinic. Laparotomy was performed the following day, and the ovum was found intact in the abdominal cavity. The uterus was firmly contracted. The rupture had taken place at the site of the old longitudinal scar The Porro operation was performed and the patient recovered.

Case XV, reported by Wyder.[22] IV-para, age 29, entered the clinic Oct. 4, 1904, in labor. The first labor ended in premature labor at the seventh month; the second labor, with transverse position and prolapsed arm, lasted three days Caesarean section was then performed. Healing was complicated by suppuration, through the abdominal wound. The third labor was premature at the seventh month. None of the children survived. The present pregnancy went to term A generally narrowed pelvis was found, and adhesion of the uterus to the abdominal wall and symphysis. Caesarean section was decided upon. About twenty hours after the onset of labor palpation revealed danger of rupture, for the fetal parts were easily to be felt. There was a difference of opinion between the woman's relatives as to the course to be pursued, necessitating telephoning, etc., and during the delay the patient experienced a severe pain, followed by prominence of the fetal parts, but with no signs of collapse. Immediately afterward laparotomy was performed. The tear, measuring 3 cm. by 1 cm., was located in the fundus, and the ovum had not yet escaped. The adhesions between the uterus and the abdominal walls were separated, as well as intestinal loops, which adhered to the site of the old scar. The tear was then enlarged to permit extraction of the child (now dead), and a Porro operation was performed. The patient made a good recovery.

Case XVI, reported by Paddock.[23] Age 36, with history of four protracted labors, three resulting in still birth, the child in the fourth labor being born alive, but dying a few weeks later of injuries received at birth. At the fortieth week of the fifth pregnancy, Caesarean section was performed for contracted pelvis, at the onset of labor. A large living child was extracted, and the uterus sutured with three layers of catgut. The patient presented herself again in the fourth month of pregnancy, and was advised to undergo the section again. At full term the patient had

severe pain, vomited and the abdomen was very tense and tender. Seventeen hours after the onset of labor the extreme abdominal distension made palpation impossible; the fetal heart could not be heard, the pulse was 90, the temperature 100° F., respiration labored. Seven hours later (twenty four? hours after rupture) the abdomen was opened; the intact ovum and blood clots were found in the peritoneal cavity and the uterus was well contracted, bleeding having ceased. The child weighed 9½ lbs. It was thought that the rupture had taken place twenty-four hours before operation, at the onset of labor, as the fetal movements which had been pronounced, ceased at that time. During the last month of pregnancy the uterus had increased greatly in size, with hydramnion. Supra-vaginal amputation was done, and the patient made a good recovery.

Case XVII, reported by Mabbott.[24] Negress, age, 23 years, Caesarean section having been performed for contracted pelvis. The fundal incision was used. The uterus had been closed with three layers of sutures, but the endometrium had not been included. The patient was seen for the first time when labor had been in progress for twenty hours. The pulse was 102, respirations 24, temperature 99.4° F. The cervix admitted two fingers, and the vertex presented above the brim. The diagnosis of rupture was not made, but when the abdominal incision was made for Caesarean section, blood and clots were found in the peritoneal cavity and the rupture was discovered. There was a laceration 7 cm. in length in the cicatrix of the fundal incision, and from this the fetal sac, about as large as the fist, protruded. A longitudinal incision, starting from the middle of the laceration, was made and a living child weighing 6¼ lbs. was removed. The ovaries and part of the tubes were removed, and the wounds in the uterus were closed with heavy chromicized catgut, the endometrium not included.

Case XVIII, reported by Schneider.[25] The patient, a V-para, with a history of difficult labors, caused by contracted pelvis, was delivered successfully by the classical Caesarean section in 1904. Two years later she was admitted to the hospital in the first stage of labor, stating that she had suffered from severe pains for seven hours. The cervix was dilated about three fingers. During the bath the membranes ruptured, and meconium-stained fluid escaped. The abdominal incision was made in the old scar, and considerable blood was found in the peritoneal cavity. The lower two-thirds of the uterine scar was perfect, but in the upper third there was a rupture about the size of a button hole, through which a portion of placenta the size of a hen's egg protruded, having been gradually forced out by strong uterine contractions. The entire scar was then incised and the child extracted in a state of deep asphyxia, from which it was revived. The patient collapsed at this juncture, and, instead of performing a Porro, as the operator had planned, he quickly sutured the wound and sterilized the patient by resecting the tubes. Later, there was a post partum hemorrhage, but the patient recovered and left the hospital with her child on the twenty-second day after operation.

Case XIX, reported by Lobenstine, to whom the thanks of the writer

are due for the history of this hitherto unreported case. First Caesarean section in 1904. Longitudinal incision in the anterior uterine wall, extending lower than my incision and not so far back in the fundus. Low abdominal incision. Second Caesarean in 1906. Short abdominal incision, entirely above

a. Umbilicus; longitudinal incision through the fundus.

b. When the uterus was sutured, the line of incision went over the fundus well to the posterior aspect of the uterus.

c. Uterus sutured in three continuous layers.

1. First layer through muscle, No. 2 catgut;
2. Second layer through peritoneum and muscle, No. 3 catgut;
3. Third, a Lembert suture, No. 2 catgut.

The recovery was uneventful. In January, 1908, the patient, when about to be confined, refused to come into the hospital. She went into labor, at term, apparently about 6 P.M. one night and apparently went into moderate collapse (after hard pains) at about 11 P.M. A physician saw her about 2 A.M. of the same night, but did not diagnosticate the condition, and left her after giving a hypodermic of morphine. She was brought to my service in the hospital in the evening of this day, *i.e.*, about 18 hours after she had been first seen by a physician. There was extreme shock and all the signs of rupture of the uterus, especially *marked* abdominal tenderness. Laparotomy was performed at once; child and placenta were found in the belly with a moderate number of clots. The uterus was ruptured from internal os up to the fundus in the median line, in one of the two previous scars. There is still some doubt as to which scar gave away. A supravaginal hysterectomy was done. The patient did finely after she had recovered from the shock and was to sit up on the tenth day, when she unfortunately developed a pneumonia and died on the seventeenth day.

Case XX. This patient was referred to me at the Post-Graduate Hospital by Dr. Brothers, and the following history was obtained: Age, 35; nativity, Russia; now pregnant for the fifth time. All previous labors had been long and difficult, with still birth in each instance, although fetal movements were observed up to the very last days. The pelvis was flat, the internal conjugate measuring 8 cm. Caesarean section was advised, and, on December 31, 1904, was performed at about full term. The usual longitudinal incision was made, and the wound was sutured in layers with chromicized catgut. The patient made a good recovery and left the hospital with her baby in good condition.

Early in August, 1906, the patient was again seen for the first time in her sixth pregnancy. She gave April 1 as the date of her last menstruation, and she appeared to be, as she herself thought, about four months pregnant. Life had not yet been felt, and the uterus appeared to be of four months size. The patient was anxious to have another child, and she was advised to have a second Caesarean operation in the latter part of December, when full term had been reached. On November, 26 at 8 P.M., a physician living near the patient was called in to see her, and he reported that the patient was having "false pains," with a slight bloody

vaginal discharge. She was advised to come into the hospital at once, and was admitted about 11 P.M. The patient was very restless and thirsty; the skin was cold; the pulse was 104, of poor quality; the respirations 44, and the cervix admitted one finger only. The abdomen was distended with fluid and the fetus could easily be palpated under the abdominal wall. Her condition grew steadily worse, and laparotomy was performed within an hour after her admission to the hospital. The fetus and placenta had passed into the abdominal cavity, which was filled with blood and clots. The child, weighing $7\frac{5}{16}$ lb. was still-born, and the patient died a few minutes after the incision was made. The rupture had occurred at the site of the old scar, but it is impossible to state when the laceration took place. There is, of course, the possibility that the patient had already reached full term one month before the estimated time, but the cervix admitted one finger only, and, if in labor at all, the accident must have occurred at the very onset.

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THE SIGNIFICANCE OF GASTROINTESTINAL TOXINS IN THE DEVELOPMENT AND MAINTENANCE OF PERNICIOUS ANEMIA.

BY LEONARD WEBER, M.D.

Professor of Medicine, New York Post-Graduate Medical School and Hospital.

During my service in the Post-Graduate Hospital, November, 1904, a patient came under our care suffering from pernicious anemia, idiopathic, so-called. After persistent entero- and hypodermocylsis with physiological salt solution, careful nutrition, etc., etc., in the management of which I enjoyed the able and pains-taking assistance of the house physician, Dr. Arthur F. Chace, the patient recovered. The case was reported by Dr. Chace, at a stated meeting of the Clinical Society of the New York Post-Graduate Medical School and Hospital, on December 15, 1905, and published in the *Post-Graduate Journal*, 1906, No. 1, p. 7.

Having had a few more cases of a similar kind since that time in private practice, it is proposed now to review the pathological and therapeutic sides of the subject. All observers are agreed that the disease is more frequent among men than women. Deducting the cases in which pregnancy, parturition, atrophy of the stomach or parasites were the prominent etiological factors, there remains a large proportion of cases in the series reported by different authors, in which the etiology of progressive blood destruction or abnormal blood production remains quite uncertain. The lemon tint of the skin in genuine cases, which in our first case was well marked; the finding of a large increase of iron in the liver by Quinke and Peters—never present in secondary anemia; the presence of urobilin in the urine, make it, indeed, quite probable that the affection is due in many cases to increased hemolysis. While some authors have held the opinion that the essence of the disease lies in defective hematogenesis, another view has been advanced in recent years by William Hunter, Grawitz, and others, to the effect that it is more often an infectious disease brought about by hemolytic toxins arising in the alimentary canal or in the oral cavity through a more or less septic state of the latter.

In two of my cases the septic condition of the mouth through carious teeth and ulcerated gums preceded the symptom-complex of pernicious anemia for a considerable period of time, and was so evident that no doubt remained in my mind about this state being the true cause of the disease. M's and one other case correspond in every particular to certain cases reported by Grawitz, in 1901 and 1902, and responded so well to the special treatment by entero- and hypodermoclysis proposed by him that, after excluding carefully all other etiological elements, we were convinced that hemolytic ferments produced in the diseased alimentary tract had given rise to pernicious anemia in these cases.

Nevertheless, there are other and probably many other cases of pernicious anemia which are not due to the destructive action of hemolytic ferments, but to abnormal or defective hematogenesis; indeed, Ehrlich's normoblasts and megaloblasts, which are found in the blood and are distinctive of this anemia, appear to point to the truth of this hypothesis. Besides, Osler and some other observers have published undoubted cases of the disease which were cured by the persistent use of Fowler's solution alone. One would be inclined to believe the etiology in these cases must have been faulty hematogenesis, corrected by the introduction of increasing doses of arsenic.

With regard to diagnosis, it will not be difficult to distinguish pernicious anemia from chlorosis and secondary anemia, because the hemoglobin in the individual red corpuscles in chlorosis is minus, while it is plus in pernicious anemia, and the average size of the erythrocytes here is larger than normal. Characteristic are the findings of megalocytes and of the large erythrocytes, Ehrlich's gigantoblasts. Poikilocytosis may be found in any severe anemia and is therefore not pathognomonic; important are also the low count of the red cells, down even to a million per cubic centimeter, the high color index and the higher percentage of mononuclears.

Our first case is one presenting all the clinical and morphological features of this disease, in which, after one year of treatment and supervision, the patient has entirely recovered and the blood returned to its normal condition. It has been established in recent years that cases of severe pernicious anemia do recover and that the number of such recoveries has increased

since the adoption of the arsenical treatment. Osler, in his Textbook on Medicine, cites four cases of this type in which the red cells fell below one million per cubic millimeter and yet the patients recovered. We have been particularly fortunate in having not only several blood examinations to confirm the diagnosis, but also in having subsequent tests made, so that we could observe the changes during the period of convalescence until the blood had returned to its normal condition. At the time of his being presented at the Clinical Society the patient was in good health and continued to be so when I last saw him about a year ago.

Case I.—P. M.; single; aged 28; policeman; born in Ireland; has been in this country ten years; entered the Post-Graduate Hospital November 8, 1904. There is no family history of tuberculosis, malignant disease or pernicious anemia, parents, one brother and one sister are living and well. Aside from measles when a child and an occasional " cold " he has always been in good health; his habits have always been good, he has used tea, coffee, tobacco and liquors in great moderation, and has not acquired any specific taint. He says that he has had two somewhat similar but milder attacks of the same disease, the first one a year ago and the second six months before entering the hospital. Six weeks before admission he felt weak and languid and had no appetite. Three weeks before admission he consulted his physician, Dr. De Forest, who subsequently sent him to the Post-Graduate Hospital. He was at that time suffering from occipital headache, pain in the epigastrium, loss of appetite, epistaxis, and general weakness. He was slightly jaundiced and ran a temperature ranging between 100° and 101° F. He had lost 25 pounds in the five months previous to this attack. On entering the hospital his chief complaints were weakness, bleeding from the nose, and soreness in the epigastrium.

Physical examination showed the patient to be about six feet tall, fairly well nourished; weight 155 pounds. His complexion was pasty and his skin of a light yellow tint. His mucous membranes were very pale. Tongue was slightly coated and moist, not tremulous. Pulse was quick, small, of fair force and low tension. The arteries are not thickened. Heart sounds were clear and no adventitious sounds were heard. At the apex of the right lung posteriorly the expiratory murmur was rather

harsh. There was an area two inches by two inches of marked tenderness just to the right of the free border of the ribs. The liver dulness extended over the following area: In the posterior axillary line six inches, mid-axillary line six inches, anterior axillary line five inches, parasternal line four inches. The superficial area of splenic dulness was five inches in length and four and a half inches in breadth. There was no edema of the extremities and the reflexes were normal. The muscles were flabby but not atrophied. Pupils were equal and reacted to light and accommodation.

During the first week the temperature ranged between 104° and 101° F. During the second week it fluctuated between 101° and normal, and through the remainder of his stay in the hospital it was about normal. The most troublesome feature that we had to deal with was persistent hemorrhages from the nose. These were so severe as to require the packing of the anterior and posterior nares. Any attempt to remove the cotton would cause the bleeding to begin anew. The hemorrhage was partially controlled by sprays of adrenalin alternating with antipyrin solution. At this time the patient complained of pain in his right ear and an examination showed petechial hemorrhages in the tympanic membrane about the short process of the malleolus.

Blood examination on admission, November 8, 1904:

Hemoglobin	55%
Erythrocytes	1,068,000
Leucocytes	4,500

Marked poikilocytosis with a number of megaloblasts and normoblasts and a few karyokinetic figures. The proportion of megaloblasts to normoblasts was about three to one.

Differential leucocyte count:

Polynuclears	60%
Small lymphocytes	26%
Large mononuclears	12%
Transitional forms	2%

The plasmodium malariæ was not present and the Widal reaction was negative.

Urine was of dark color and apparently contained a large amount of coloring matter, such as urobilin. The diazo-reaction

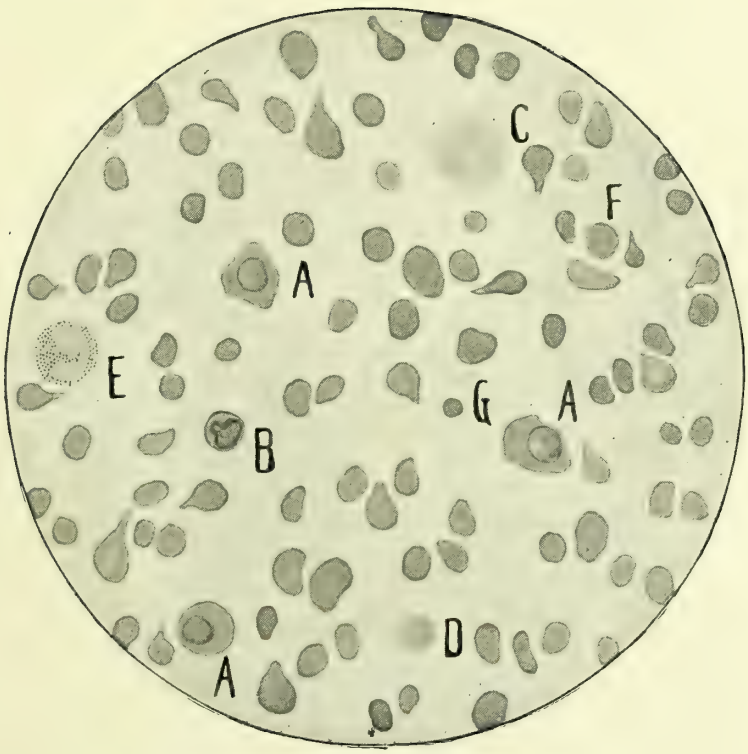

FIG. 1.—A, megoblast; B, normoblasts; C, polynuclear; D, small lymphocyte; E, eosinophile; F, poikilocytes; G, microcyte.

(Weber)

was negative. No leucin or tyrosin crystals were found. When the temperature was at its height there was a trace of albumin and a few hyalin and finely granular casts found. On leaving the hospital the patient's urine was normal. Repeated examinations of the sputum showed the absence of tubercle bacilli. Several examinations of the feces failed to discover any parasites. The ova of anchylostoma duodenale and the bothriocephalous latus were especially looked for.

A week later the hemoglobin was 50% and the red cells had dropped to 788,000 and the white cells were 7,000. The plate on preceding page is a composite drawing from a preparation of the blood made at this time by Dr. B. F. Cline, of the laboratory.

When this blood count was taken the patient was so completely exhausted that death seemed imminent and his relatives were notified to come to the hospital. In addition to daily high enteroclyses of a liter of normal saline, he was given hypodermoclyses of 300 c.c. of 0.9% saline solution in the pectoral region twice a day for three days. These seemed to cause the turning point in the disease and from this time on he continued to improve. The patient's daily diet consisted of three ounces of beef juice, scraped beef sandwiches, bread, cereals, custard, broths, egg-nogs, lemon, albumin-water and, after the first week, a few green vegetables. He was given Fowler's solution, five drops three times a day, the dose being increased one drop daily until the patient was receiving 16 drops three times a day. He was given also one half an ounce of hemaboloids three times a day, and while he was running a temperature 10 grains of quinin muriate twice a day.

He improved very rapidly and on November 28 his hemoglobin had reached 80% and his red cells had risen to one million and a quarter per cubic millimeter. A differential leucocyte count showed 40% of small lymphocytes which is rather characteristic of this disease. The poikilocytosis was just as marked as at the former count and there were a large number of megaloblasts and normoblasts present.

He left the hospital on December 19 in fairly good condition although his blood count was practically the same as that of November 28. The treatment was continued after leaving the hospital and a blood examination taken April, 1905, showed marked poikilocytosis and a large number of megaloblasts and

normoblasts. Last summer he obtained a position in the country where he spent most of his time out of doors. He improved to such an extent that he gained 40 pounds in weight and he was able to resume his duties on the police force last fall. He feels perfectly well now, and a blood count taken a few weeks ago, just one year since he entered the hospital, showed the following results:

Hemoglobin	85%
Erythrocytes	4,320,000
Leucocytes	5,200

The red cells were perfectly normal in size and in shape, and repeated examinations failed to show any normoblasts or megaloblasts.

Differential leucocyte count:

Polynuclears	72%
Small lymphocytes	21%
Large mononuclears	5%
Eosinophiles	2%

As to the completeness of the recovery, I think the blood count, the patient's feelings, and his appearance will dispel all doubts. As to the diagnosis of an idiopathic anemia, the patient's history previous to the attack, the negative results after careful examinations of the blood, sputum, urine, and feces during the attack and his subsequent history all failed to show a cause for the anemia. The clinical picture of the case with its insidious onset, the extreme pallor of the patient without any emaciation, and the great exhaustion were very suggestive of the disease before the blood count had been taken. The blood examination with the marked oligocythemia, the relatively high color index, the extreme poikilocytosis, the large number of megaloblasts and normoblasts and the lymphocytosis is pathognomonic of pernicious anemia.

Case II.—L. M., 32 years, German, single, mechanic. No hereditary nor acquired taint. Had never been strong and vigorous but had always been able to work at his job in a piano factory. From his twenty-fifth year on troubled with dyspepsia and more or less obstinate constipation; he had also occasional diarrhea and slight epistaxis. Early in 1905 he had to give up work, and coming under my observation in March he presented

the characteristic lemon tint, pale mucous membranes, flabby musculature, foul breath, and a few petechiæ here and there and some upon the abdomen; spleen somewhat enlarged; no temperature. Blood examination showed poikilocytosis, some normoblasts and a few megaloblasts, red cells about two million to the cubic centimeter. Although characteristic enough, the pathological state of his blood was much less marked than in the case of P. M.

The history of the case was carefully gone into, but with regard to its etiology nothing remained but the supposition that gastrointestinal ferments might have caused hemolysis and blood degeneration.

This case of pernicious anemia being yet in the early stage and comparatively light the patient's stomach and bowels were first thoroughly irrigated and cleansed and then enteroclysis of normal salt solution of between one and two quarts in the twenty-four hours applied for several days. Fowler's solution was also given in gradually increasing doses, proper diet, warm baths, and good nursing ordered, and the patient treated in his own house. Improvement followed quickly and final recovery took place in the course of the summer. At the end of 1905 the blood count was about normal and has remained so.

Case III.—Mary S., age 26 years, single, Irish, servant. No history. For about five years decaying teeth and ulcerating gums with occasional bleeding from them. Had been in hospital for this but failed to have her teeth and gums attended to after being discharged. When she came under my care three years ago she was able to be about but had bled rather profusely from the mouth for several days. An examination disclosed extensive decay of teeth and diffuse ulceration of gums. Blood count showed only two million red cells, some poikilocytosis, but neither megalo-nor normoblasts. I took especial care to have her treated by a competent dentist, and had then but little difficulty in restoring her gums to normal condition by appropriate local treatment. After the administration of iron and arsenic and a three months' sojourn in the country the patient was completely cured.

Case IV.—Louisa F., aged 30, American, single, teacher, a pale, poorly nourished woman, presented herself in October, 1906. She had never been robust, suffered much from headache

and of late of general weakness and had spit up a little blood the day before she called, which she said came from her mouth and throat. When I examined her mouth I found most of her teeth bad and the gums extensively ulcerated, and I urged her to go at once to a dentist and return for further examination and treatment as soon as he was through with her. The patient failed to return; but in January, 1907, I was sent for and found her lying in bed in a miserable and badly ventilated basement dwelling, very ill indeed, bleeding profusely and very persistently from swollen and ulcerated gums. Unfortunately she had not followed previous advice but drifted along until she had to go to bed in an exhausted condition. A hurried examination of her blood showed great diminution of the erythrocytes, poikilocytosis and numerous lymphocytes. In spite of strenuous efforts made by myself and assistant and nurse we did not succeed in controlling the bleeding or in overcoming the general asthenia in which we found her, and she died from sepsis and exhaustion at the end of two weeks.

The proportion of cases of pernicious anemia caused by cytolytic ferments originating through diseases of the oral cavity or the gastrointestinal tract may not be as large as some of us think or believe, but that quite a few cases have that origin is certain from the description of the above cases and the observations of many other authors. And that such cases can be greatly benefited and even cured by treatment as above described and carried out assiduously and persistently does not admit of any further doubt.

BIOLOGY AS THE BASIC PRINCIPLE IN INFANT FEEDING.

BY HENRY DWIGHT CHAPIN, M.D.

Professor of Medicine (Diseases of Children) New York Post-Graduate Medical School and Hospital.

Anyone looking over the teachings of American and European pediatricians concerning the artificial nutrition of infants, can hardly fail to conclude that this subject is far from being established on a scientific basis, because of the conflicting theories in regard to the proper methods of selecting and preparing the food. Upon looking below the surface, it will be found that not a little of the conflict of opinion concerns details merely, and is due to differing conditions in the various countries; what is considered as being absolutely essential in one place seems to be of little consequence in another, so that the stress laid upon the particular procedure appears incomprehensible; and also to the fact that no basic principles appear to have been recognized by all.

It seems almost unnecessary to state that infants are essentially the same in all parts of the world, especially in the temperate zones, where the great mass of population is found, and therefore must have the basic nutritional factors in common. Their differences, then, must be due to individual idiosyncrasies and difference in environment.

It is manifestly impossible to establish any narrow principles adapted to every nutritional peculiarity of every infant under every kind of environment; and it might as well be accepted, first as last, that there are no fixed quantities or forms of food that are invariably needed by each infant under any or all conditions. We must look further for basic principles, the more so because it is well known that no two individuals are exactly alike, and Nature has endowed every living thing with more or less power or ability to change its methods of living and nutrition as its environment and food-supply change.

The infant possesses the power of adapting itself to many apparently unnatural methods of nutrition and forms of food, and it is this fact that accounts for the many different infants'

foods and methods of feeding which have been more or less successfully employed and advocated, and also for the belief held by some that there is no science in artificial infant feeding, each infant being a law unto itself.

The only principles which can be universally accepted are those that apply to all infants, no matter what their environment may be, and it will greatly advance the cause of scientific infant nutrition if the fundamental facts on which these principles are based and the principles themselves can be clearly enunciated and accepted. The application of these principles will have to be made according to conditions as they are found and will constitute the *art* of infant feeding. In the application of the fundamental principles, each pediatrist will have perfect freedom; yet the same scientific principles will be recognized and applied systematically all over the world and results can then be compared intelligently. With the fundamentals removed from the field of discussion, valuable lessons can be learned from comparing the results of the different methods of applying the basic principles, and real progress will be made. As it is to-day, many comparisons of methods of feeding are misleading and unreliable, because, in numerous instances, different principles are being applied and all of the factors involved are not taken into consideration.

It will be helpful to see how much can be taken from the realm of discussion and just what points lie within its domain.

It is self-evident that the infant belongs to the animal kingdom and, therefore, is subject to all the general laws of animal life. Now, it has been determined that all animals begin their existence as a single cell, which passes through various forms of increasing complexity to reach the adult state. There are no sudden breaks in the process of development. It is continuous. In the early periods of growth all higher animals are so much alike in form as to be indistinguishable, and it is only when development has proceeded to a marked extent that specific differences appear.

The food of all animals is derived primarily from the vegetable kingdom. Some animals are exclusively vegetable feeders, others subsist on vegetable and animal food, while still others are strictly flesh eaters. Hence, the natural division of the animal kingdom into the herbivera, omnivora and carnivora.

The young of all these groups of animals derive nourishment from the parent's body until able to subsist on the parent's food. When one studies the physiology and physiological chemistry of all the different classes of animals, old and young, it is found that the bodies of *all* are composed essentially of the same materials; they *all* require and obtain essentially the same elementary food substances, and *all* have digestive juices which will digest them. So far, there is a unity in the animal kingdom. While the food of animals, old and young, shows a wide diversity of form, it has been found that their components fall into a few broad classes. (1) The protein substances, which are distinguished by containing nitrogen; (2) mineral substances; (3) fats; (4) carbohydrates; (5) water. Each of these divisions, except water, comprises a genus including many specific forms. For instance, under protein there is classed a large number of substances that differ in form, taste, chemical composition and physical properties, their only point in common being that they contain nitrogen. Fats include butter, fat of meat and fish, olive and other edible oils; carbohydrates comprise cellulose, starch, dextrin, maltose, glucose, cane-sugar and milk-sugar.

The elementary food substances are not met with in Nature in an isolated state, but mixed together in complex combinations and in a great variety of forms, such as hay, grass, mosses, wheat, oats, corn, barley, lettuce, spinach, celery, turnips, potatoes, bread, milk, eggs, fish and meats, sugars, molasses and syrup. It would be impossible to feed a dog on cut hay, or a cow on bones and meat. It might be proved by chemical investigation that there was enough proteid in the hay for the maintenance of the dog and that it would produce enough calories to meet all of its requirements. The same might be proved to be true of the meat and bones given to the cow. Yet, it is plain that such a method of establishing the suitability of food is wrong, and that foods may be chemically right but practically all wrong. Many of the teachings concerning infant feeding have been along this line, and their principal effect has been to make very complicated what should be a simple matter. It will be seen, then, that when it comes to selecting the food for any species of animal, the form and physical condition of the food assume paramount importance.

The great nutritional difference between animals of various species is in the form of the food and manner in which they secure and digest it; not that they require different food elements. Everyone is familiar with most of the gross differences in form of the commoner animals and thoroughly appreciates the reasons for the webbed feet of the water fowls, the hooked beaks of birds of prey and the large canine teeth of dogs and cats. Yet, comparatively few realize that there are as wide differences in the form of the digestive organs of different species of animals as in their external shapes or appreciate their significance. In selecting food for any animal, the first thing is to see that it is adapted to the animal's digestive apparatus, and then look into its nutritional value. It has also to be borne in mind that animals of the same great class have different types of digestive organs which are not equally efficient in digesting the same kinds of food. For instance, it is well known that the horse can not digest hay or grass as thoroughly as the cow, because of its relatively small stomach which empties food into the intestines rapidly, while the cow's stomach is large and retains the food longer. Also, that individuals of the same species have wide differences in efficiency of digestion and assimilation.

What has been stated thus far is so generally accepted by scientists as not to need further discussion and clearly shows that the great nutritional differences between animals are *biological* differences.

In the earlier stages of development, the young of herbivorous, omniverous and carniverous mammals are nourished in the same manner through a chorion and placenta; but when birth takes place, the development and divergence of the digestive tracts commence. Then, a wide change in the physical properties of the food is found; in other words, while the mammary glands of all species secrete milk that is composed of the five generic divisions of food, viz., proteids, salts, fats, carbohydrates and water, the physical properties of these elements are not the same in all milks. The differences in the digestion of food that characterize the herbivera, omnivera and carnivora in adult life have their beginning while the young are still nourished by the mother. The young of all of these classes of animals have digestive secretions that produce, as far as is known,

essentially the same chemical changes in the same kind of food during digestion. As far as chemical transformation of the food elements are concerned, there seems to be a unity in all animals, young and old; but when the development of the digestive organs in which these chemical transformations take place is considered, it is found that, at the start, the digestive organs of all are very much alike. As the young animals grow, their digestive organs diverge widely in form and method of operation, until the vast differences found in the adults of the different classes of animals result.

The stomach in most animals is rudimentary at birth and develops rapidly while receiving mother's milk; after natural weaning it has the form and function of the adult stomach of the species. It is known that milks of the different species are curded or clotted by the action of the gastric secretion of young animals, or by pepsin and acid, and it will be found by a study of comparative anatomy and physiology, that the mother's milk clots in a form that will act in developing the digestive tract of her young and hence in a form similar to that of the food in the fully developed digestive apparatus of the adult. Development of the digestive organs is as continuous as is the development of the exterior parts of the body. The digestive organs are not suddenly made over at weaning.

It is idle to discuss whether the elements of one milk are more nutritious than those of another kind. The proteids, salts, fats, and carbohydrates of all milks are equally nutritious beyond a doubt, for the young of all species develop equally well on their mother's milk. It is only when the attempt is made to supply the food of one species to another that the question of nutritive value arises. As was stated before, it would be impossible to feed hay to a dog or meat and bones to a cow, but this same general principle is being applied when the milk of one species is fed to the young of another species, and the reason that it is not more disastrous is that the divergence in character of the food during the suckling period is not very great, and that the change in the solidity or denseness of the clotting of the milk in the stomach is regulated by the young animal's digestive secretions. By careful training, the stomach is taught to produce a secretion that will not form a too unnatural clot. The tendency of the animal to

adapt itself to its environment and food asserts itself. Here, also, it is seen that the great nutritional differences between young animals are not chemical but biological. The same laws hold for adult and young.

With the fact in mind that all animals, adult and young, are built of the same materials and require the same general food elements to maintain their existence, although in different proportions, how meaningless it is to state that one of the first essentials in selecting a food for infants is to see that it contains the same ingredients as human milk, viz., proteids, salts, fats, carbohydrates and water, as if these ingredients were peculiar to human milk, and were not found in the food of all animals. The emphasis that has been laid on the " same ingredients as mother's milk ", meaning the generic divisions of all foods, has given an erroneous opinion to students who have not studied the general science of nutrition and has confused them. It is not uncommon to find young men, fresh from the medical schools, who believe that salts of food mean ordinary table salt, and a study of many of the text-books on children will show that some of the writers themselves have not a clear idea of the science of nutrition. A large number of authors have treated proteids, salts, fats and carbohydrates as if they were specific, well-defined entities, instead of large genera. After laying down a theory, they sometimes abandon it in practice, and no amount of fitting can make their practice conform to their theory.

The one thing that distinguishes suckling animals from adults, is the form in which their food is supplied. It contains the same elements as the food of the adults contains, classing them as proteids, salts, fats and carbohydrates. Generically, they are alike; specifically, they differ. The fat of milk is a mixture of several fats; the salts do not exist separately or as a single one, but in combination with the proteids; the proteids assume two forms, principally calcium casein and albumin. It is claimed there are at least two forms of milk-sugar. When it comes to dealing with caseins, it is found that a whole group of casein compounds exists, having differing properties, and many of these forms are used in infant feeding.

How can there be any scientific comparison of results in feeding infants unless these differences are recognized and the specific

form of proteids and carbohydrates employed particularly be mentioned?

In practice there are used:

Plain, fresh cow's milk
" " " " diluted with water
" " " " " " gruels
" " " " " " " dextrinized

Fresh cow's milk and lime water
" " " " potassium carbonate
" " " " sodium bicarbonate
" " " " sodium citrate

Cow's milk or cream with whey
" " " " " " and alkali

Cow's milk and acids—precipitated milk

Cow's milk and acid bacteria—precipitated milk and bacteria.

In these different methods of furnishing cow's milk to infants, about seven different forms of proteids are employed; but this fact is not generally recognized and makes teaching highly empirical.

The attempt at establishing a calorie basis of feeding exclusively is based on a mistaken idea. The calorie is a measure of heat and energy. The infant is not a machine for principally turning food into work. A certain amount of work it has to do in growing and in carrying on its bodily functions, but its *great* demand is for food that can be converted into *body tissue*.

Condensed milk as often fed to infants has a high value in calories because of the excess of sugar it contains. Condensed-milk babies are, as a rule, fat but poorly nourished; they have stored up calories as fat, but proper growth and nutrition calls principally for storage of proteids and salts.

SUMMARY.

The details of the different manipulations required to make various milks acceptable to the infant's stomach constitute the *art* of infant feeding. All these details, however, to be of lasting benefit, must be in accordance with biological laws, and, in a last analysis, biology will decide both the possibilities and limitations of every method that is advanced. It is the only lasting and authoritative standard to which we may bring all

theory and practice of infant feeding. Thus, the advancing science of biology will put in order and systematize our knowledge on this as on so many other vital subjects. Surely, no other subject is more important than the proper nutrition and growth of the infant. As an example, we have shown that the proteids of the various milks have *developmental* as well as nutritive functions. The former function must be considered as well as the latter. As the proteids of all milks change into solids in the stomach of the young animal in a form analogous to the food of the adult animal—the object being to develop the motility of the stomach and digestive tracts for maturer work—we at once see that, from a biological standpoint, the milks of different species are not interchangeable. The mere computation of the proteid content of a certain milk is not sufficient. We must insist that, as nearly as possible, it shall take the same form as in the natural milk of the animal to which it is given.

Mere soluble proteids, although possibly satisfactory from the chemical standpoint, will not fulfil the biological necessity and, hence, will not prove adequate for prolonged exclusive use. This is an example of how important it is to know the biology as well as the chemistry of milk. Since it is the curd (nucleoproteid) that builds up tissue, the clinical results bear out what a knowledge of biology foretells. A recognition of the structure and function of the various digestive tracts in connection with the milk early furnished is, likewise, necessary to complete our knowledge of the biology of the subject.

For some years the writer has studied and considered infant feeding from this standpoint, and by his teachings and writings has tried to make this method understood and followed by the profession. The articles are comprised in the following bibliography:

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THE NEED OF POST-GRADUATE INSTRUCTION IN PEDIATRICS.

BY AUGUSTUS CAILLÉ, M.D.
Professor of Medicine (Diseases of Children), New York Post-Graduate Medical School and Hospital.

In the making of a medical practitioner there is still a lack of system and uniformity, which is difficult to explain except as we view it as one of the forms of inherited or transmitted human inertia noticeable along sanitary lines in general.

At the present time the best results are probably obtained where a student, endowed with good "preliminaries," enters a medical school which is part and parcel of a large university so located as to be able to offer to the senior student in medicine ample hospital facilities and abundant clinical material; with its curriculum so arranged that the undergraduates' time is not taken up by didactic lectures, and the student is early brought into contact with the evidence of nature's manifestations in health and disease by means of practical courses in chemical, physiological and pathological laboratories.

Small medical colleges can, no doubt, also turn out good practitioners on account of the greater care given to each student, but to accomplish this they must offer a system of "object lesson teaching" by means of which, as Agassiz tersely says, "One may study nature, not books."

Faulty as it is, we must confess that the wholesome impetus of a good medical school makes the student eager for the two years' hospital and bedside training which is to follow, and at the end of which he is fairly well equipped to be let loose upon the public as a general practitioner.

In the realm of pediatrics, however, the young physician as he starts out upon his life of service, is deplorably ignorant. He may be able successfully to cope with difficulties in diagnosis and to formulate indications for treatment and even carry out proper treatment in the adult, but when he is confronted with the problems presenting themselves as regards the hygienic, dietetic and specific management of the ailing child, he stands, as far as practical knowledge goes, "*vis à vis de rien*," and is unable to stake and prove his claim in pediatrics

unless he has had the very exceptional good fortune to have spent half a year in a hospital devoted to diseases in children.

During a twenty years' service as teacher in pediatrics, the writer has not met a single young hospital graduate other than ex-internes of a children's hospital, who, on inquiry, did not freely admit his lack of practical knowledge regarding a line of professional work which makes up one half the practice of the family physician. It is evident, therefore, that the facilities for acquiring the knowledge necessary to combat preventable infantile sickness and mortality are totally inadequate and that more practical instruction along the lines indicated is urgently needed.

How best to meet the demand for practical instruction in pediatrics has taxed the efforts of those devoted to this kind of work.

The value of a good post-graduate course lies in the fact that under guidance of competent teachers one may first see a patient in the "*dispensary,*" then in the *hospital ward,* where medical, operative or specialistic treatment is carried out in detail. One may examine the patient, study his "chart," examine blood, sputum, excreta, puncture fluids in the *laboratory,* and if the patient dies, one may witness the autopsy and examine the specimens obtained.

Dispensary Clinics.—A large ambulatory service is the *sine qua non* feature of a good medical school. Dispensary work (apart from its value to the community) is excellent discipline for acquiring the art of making more or less accurate diagnoses and sharpening the power of observation.

Hospital Wards in Connection with the Clinical Laboratory.—The "wards" in connection with a clinical laboratory afford every facility for accurate study and proper treatment of patients. The course and termination of disease may be watched, and it is frequently possible to "prove the case" by transillumination, surgical procedure, or autopsy. The "wards" are available for illustrating and demonstrating methods of prophylaxis (ventilation, isolation, disinfection and protective inoculation), for diet kitchen work, palatable medication, for surgical, orthopedic and other manipulative or electrical therapy and for teaching physical diagnosis and diagnostic punctures.

Bedside Instruction.—Making rounds with a *large class of students* is of little value. To make bedside observation available

for large classes, any one of the class should be allowed to visit the wards and examine patients and study their records during a set time of two or three hours daily and at a time in which there will be no serious interference with other clinics or other work. For purposes of bedside instruction the writer makes use of a diagnosis chart which assists a student to go through a routine examination of any case assigned to him.

Amphitheatre clinics have their advantages and drawbacks. To utilize clinical material to the best possible advantage requires a large floor space for setting up ten to twelve tables for the simultaneous examination of as many patients. Demonstrable regionary lesions can be marked in colors on the skin of a patient or may be recorded by means of a chart. Not more than four matriculates should be invited to each table and, guided by competent instructors, should be given an opportunity to examine each case. After such demonstration *en masse* the students would take their seats and the teacher would proceed to point out the important differential diagnostic features and therapeutic indications in each case. Diagnostic punctures and manual methods of treatment, such as stomach and bowel irrigation, bathing, feeding by gavage, tapping the thorax, the abdomen, the spinal canal, intubation, removal of adenoids and tonsils, etc., can be demonstrated with advantage before a large class.

The broadening influence of pediatric study has not been sufficiently emphasized and is probably underrated, but must be conceded as we realize how thoroughly the practice in diseases of children brings us into close touch with almost every other special line of medical work, including general surgery, the eye, ear, nose, and throat specialties, orthopedics, skin diseases, etc.

Owing to the lack of flexibility in medical matters much time will pass before medical schools will be properly equipped to teach practical pediatrics, and thus the urgent need of post-graduate facilities for pediatric study is given.

It is so ordained that all evils will in time work their own cure, and the few reflections here penned are offered as a small contribution to the propaganda of adjustment and as a tribute to the genius and perseverance of Dr. D. B. St. John Roosa, whose pioneer work in behalf of post-graduate instruction in medicine, merits our admiration.

PARTIAL THYROIDECTOMY COMBINED WITH ROENTGEN TREATMENT IN BASEDOW'S DISEASE.

BY CARL BECK, M.D.

Professor of Surgery, New York Post-Graduate Medical School and Hospital, etc.

The marvellous results of surgical treatment of tachycardia strumosa exophthalmica, called Basedow's disease, offer convincing proof of the correctness of the theory that the etiologic factor in this mysterious affection is based upon chemical disturbance, in other cases upon a pathologic or excessive secretion of the thyroid gland, producing intoxication of the body, especially of the central nervous system.

The statistics of Kocher, who performed excision of the thyroid gland in two hundred cases of Basedow's disease, speak more forcibly than any clinical or anatomical observation. There is only one drawback to the operative treatment, namely, the mortality-rate, which, even in the skillful hands of Kocher, yields $4\frac{1}{2}$ per cent., while some of our best surgeons admit as high a rate as 30 per cent.

Four years ago I performed thyroidectomy upon a married woman, 48 years of age, at the Post-Graduate Hospital. She complained of palpitation of a most distressing character, temporary dyspnea, diarrhea and general exhaustion. There was considerable enlargement of the thyroid gland, while exophthalmos on both sides was little marked (Fig. 1). Tachycardia was intense, the pulse varying between 120 and 160. A systolic murmur and tremor of both hands could also be observed. The alarming condition of the pulse forbade the administration of a general anesthetic from the beginning. Therefore, hypodermatic injection of $\frac{1}{6}$ gr. of morphine, followed by the infiltration method of Schleich, was employed. A bilateral incision was made around the lower configuration of the tumor, which could be removed with the loss of a moderate amount of blood. A small portion of the right lobe was left. The pulse was 162 pro minute from the outset, while at the end of the operation it had risen to 168, the volume being somewhat weaker. After an hour of continuous stimulation (subcutaneous saline infusions and injections of camphorated oil), the patient rallied,

the pulse became stronger and slower (140) and the general impression received seemed to indicate a favorable outcome. After twenty-four hours symptoms of toxic myocarditis became marked, respiration frequent and shallow, the pulse disappeared and the patient succumbed under cyanotic symptoms. There was no secondary hemorrhage. The extirpated lobes (Fig. 2) showed irregularly dilated follicles filled with colloid material and were lined with cubical epithelium.

The fact that even under the most unfavorable circumstances the operation could be carried out rapidly and with comparative ease, as well as the post-operative course, point to the liberation of a toxic *imponderabilium* by manipulations during operation, and possibly elimination of too large an amount of thyroid tissue. Therefore, it seemed to me to be important in the first place that all manipulation should be as delicate as possible, force especially to be avoided. Besides, it was evident to me that the gravity of the interference would be lessened by removing only the largest lobe, which would prevent the other extreme: underthyroidosis.

The observation that the Roentgen method has a specific influence of a shrinking nature on the walls of the blood vessels, which can be utilized in succulent tumors, induced me to try the rays in mild cases of Basedow's disease with the most gratifying results. Encouraged by these, I began to apply the Roentgen method as a secondary means of therapy for the remaining lobe.

I carried this theoretical deliberation out in practice four years ago in a very severe case of Basedow's disease (Fig. 3), the history of which is published in my report: "Ueber die Combination von Excisions- und Roentgen-Therapie bei Morbus Basedowi."[1] Fig. 4 shows the patient three years afterwards in perfect health, a slight trace of exophthalmos still being present, which has since disappeared entirely. It was remarkable how quickly the remaining lobe disappeared under the post-operative Roentgen treatment.

One of the most useful features of the Roentgen treatment in this instance is that the gland can be reduced gradually, so that the reduction is virtually regulated by the operator, whose instinct will lead him to avoid overthyroidosis as well as underthyroidosis. These results were corroborated by the observa-

1. *Berliner klinische Wochenschrift*, 1905, No. 20.

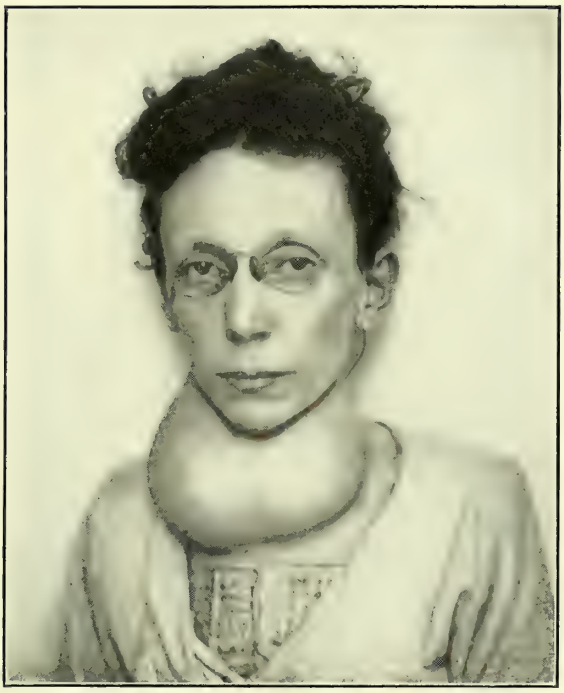


FIG. 1.

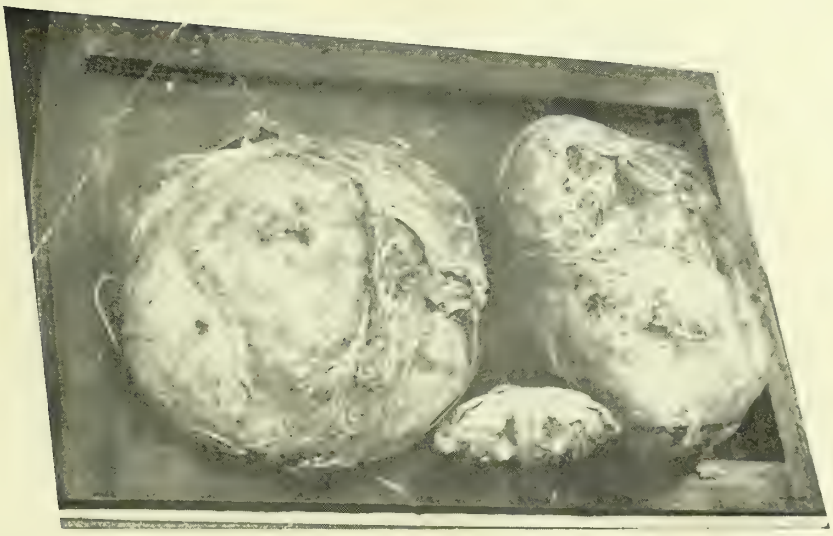


FIG. 2. (Beck)

FIG. 3.

FIG. 4. (Beck)

FIG. 5

FIG. 6. (Beck)

tions of Gilmer, Schlesinger, Stegmann, Sklodowski, Schwarz and Freund.[2] Since that time I have treated fifteen cases of Basedow's disease by ordinary Roentgen treatment as well as by the combination method.

In one of these cases toxic signs resembling the symptoms of a severe pneumonia sometimes persisted at various intervals for two weeks after removal of the right thyroid lobe. The patient, an Italian girl, seventeen years of age, (Fig. 5), complained of great respiratory disturbances, palpitation, tremor and a sensation of heat over the skin of the body; excessive perspiration, restlessness at night and general decrepitude.

The enlargement of the thyroid gland was considerable, while the exophthalmos was of moderate degree. The pulse varied between 120 and 150. The signs of Moebius and of Graefe were well marked, and the knee reflex increased. Extirpation of the large right lobe was performed in the same manner as described above, the loss of blood being excessive on account of the immense vascularity of the external capsule. The pulse after the operation was 158, but fell below 130 during the first ten days thereafter. The respiration was sometimes stertorous and reached 70 pro minute at irregular intervals. For fully twelve days more or less dyspnea was present. No change of note was perceived on percussion, but auscultation revealed extensive rales, and sometimes tracheal whistling, as in asthmatics, could be heard from a distance. That there was not the slightest wound disturbance could be proven after recovery by the fine linear scar in the incision line. The patient received only six post-operative exposures. Nearly a year has elapsed now since she regained perfect health.

In contrast to these last two cases, in which considerable enlargement was combined with only a slight degree of exophthalmos, while the other characteristic symptoms were well marked, I mention two young women in whom the opposite condition was observed. Difficult as it is to explain this phenomenon satisfactorily, it points to a difference of the composition of the thyroid secretion. It is not be the quantity alone, but the quality of the product, which determines the different relations of the size of the gland to the various degrees of exophthalmos. In other words, there must be a condition

2. *Muenchener medicinische Wochenschrift*, April 23, 1907.

of disthyroidosis besides that of over- and under-thyroidosis, which causes an underfined and indirect influence upon other organs of internal secretion. The case illustrated by Fig. 6, was characterized by a very moderate enlargement of the thyroid gland, while the exophthalmos was considerable. Tremor and tachycardia were very marked. Roentgen treatment was administered every second day during the first week, and twice a week thereafter, until four weeks after the beginning of this therapy a slight dermatitis could be noticed. The treatment was then suspended. In the meantime the swelling of the thyroid gland had so far subsided that it could not be palpated. All subjective symptoms disappeared and the patient felt perfectly normal, but the exophthalmos still persisted. For this reason the local treatment was again employed for another two weeks. Then the exophthalmos gradually disappeared.

The case illustrated by Fig. 7, showed a still smaller swelling of the thyroid gland, while the exophthalmos was more marked than in any of my other cases. Tremor and tachycardia had reached a high degree. Dyspnea, tremor and general uneasiness became more aggravated when the patient, a woman twenty-five years of age, became pregnant. It was only after the tenth exposure that the tumor became smaller and the subjective symptoms milder. After the eighteenth exposure the swelling disappeared, while the exophthalmos had lost very little of its marked features. At the present time the pulse is 84 pro minute and the general condition of the patient tolerable. A further report of this case will be given in the near future.

The most remarkable of my cases is that of a boy, eleven years of age, presented cured to the New York German Medical Society, November 4, 1907. The delicate patient was born in Hungary and descended from ill-nourished parents. The case came under my observation eighteen months ago, when he suffered from palpitation and tremor and showed a soft thyroid enlargement the size of his fist. Exophthalmos was considerable, Fig. 8. The tachycardia was marked, the pulse 170 pro minute, occasionally even 186. At first the patient was irradiated every second day during the first two weeks, then every third day, until five weeks after the beginning a slight dermatitis made interruption of the Roentgen therapy necessary. For two weeks during that period the tumor decreased considerably. Irradia-

FIG. 7. (Beck)

FIG. 8.

FIG. 9. (Beck)

tion was again applied once a week, until four months after the beginning of the treatment the tumor as well as the tachycardia had disappeared. The exophthalmos had decreased, but a trace of it can still be perceived at the present time, Fig. 9. Otherwise the patient is absolutely normal. In this case no medication except the Roentgen treatment was given. In view of the indigent circumstances of the family, dietetic measures could not be observed. Just as in tuberculosis, financial considerations unfortunately play an important part.

Of the remaining eight cases only one failure has thus far been observed; but even here the most distressing sensation of uneasiness on the part of the patient, a girl of twenty-two years, disappeared, while the thyroid enlargement and the exophthalmos showed no reaction. The tumor in this case was harder than in the other patients.

I made it a general rule to administer the rays through my tubular diaphragm, using considerable energy with soft tubes. Irradiation lasted five minutes and was repeated every second day during the first and, as a rule, every third day during the following weeks, until there was either a marked decrease or a dermatitis reaction. After the latter had ceased, irradiation was again employed about twice a week, until complete disappearance or a new reaction. All patients, with the exception of the boy, whose case is described above, received fairly large doses of Fowler's solution, up to twenty drops three times a day, provided the digestion was not disturbed, in which case it was temporarily discontinued. The diet was carefully controlled.

On the basis of these observations I feel justified in suggesting:

1. Slight thyroid enlargement in Basedow's disease should be treated by the Roentgen diaphragm-method at short intervals.

2. Large Basedow goitre should be treated by the combination method, namely, the larger lobe is to be removed under local anesthesia and without the use of antiseptics of any kind; the other portion to be irradiated as soon as the reaction from the operation is over.

3. In advanced cases of Basedow's disease, where alarming symptoms forbid immediate operative interference, Roentgen treatment should precede the operation until improvement.

REPORT OF A CASE OF NARROWING AND CONSTANT PATENCY OF THE PYLORUS.

BY MAX EINHORN, M.D.

Professor of Medicine, New York Post-Graduate Medical School, and Hospital.

At first sight it appears rather peculiar that opposite conditions, as stenosis and patency of the pylorus, should be found in one and the same patient. It probably occurs, however, relatively often. I shall first describe such a case and then add a few remarks.

Dec. 27, 1907. Wm. J. B., 50 years old, has been suffering for the last five or six years from dyspeptic attacks, lasting usually one to two weeks, and consisting only of pains one to two hours after meals. Two years ago his trouble increased and the attacks lasted a longer time (three or four weeks). Pronounced constipation developed. Six months ago the patient began to vomit more frequently and he was suffering almost constantly. At times the patient felt severe pain in the upper epigastrium, sometimes only a feeling of pressure in the stomach and burning in the scrobiculus. Vomiting now ensued regularly every day or every other day, a large quantity of fluid and food being ejected. Blood was never found. His sleep was often disturbed by attacks of pain. The patient lost steadily in weight, about 50 pounds in the last six months. The patient has always used alcohol and tobacco freely. He denies a history of syphilis.

Present condition. Thoracic organs are apparently normal. Stomach extends to two fingers above the navel. Clapotage can be elicited in the left hypochondrium somewhat higher than normal. The liver dulness begins in the right mammillary line, one finger's width below the mammilla, extending two to three fingers' width beyond the right costal arch.

Examination of the stomach contents revealed the presence of food from the day before, the presence of HCl, some bile, and a moderate acidity. Sarcinæ were not found. A few days later the patient was examined in the fasting condition after having eaten a plate of rice with milk on the previous evening. Examination with the stomach tube brought up about 300 cc. of

greenish contents in which much rice and other food remnants were found. Free HCl was 60, total acidity 80; benzidin test revealed presence of occult blood.

Urine is free from sugar and albumin.

The blood showed 5,100,000 red, 8,600 white blood cells, hemoglobin 85%.

The diagnosis of pyloric stenosis, probably of a benign nature, and beginning cirrhosis of the liver, was made. The patient was placed on a strictly liquid diet and the stomach was washed daily in the fasting condition. Medicinally he received magnesia usta and bismuth subnitrate, and sajodin.

The patient felt much better, the vomiting ceased and the pains disappeared; he slept well. Examination with the stomach tube, however, revealed every morning the presence of 200 to 600 cc. of greenish contents, containing plenty of food remnants. HCl and bile were always present; the acidity was between 60 to 80, occult blood was almost constantly found.

Occasionally peristaltic restlessness of the stomach could be plainly seen in the upper left epigastric region.

On Jan. 2, 1908, two beads, filled with methylene-blue and coated with mutton tallow, were left in the stomach for four and a half hours. The length of the thread of one of the beads was 50 cm., that of the other 75 cm. On withdrawing the beads it was observed that they both contained the methylene-blue and tallow; therefore it was concluded that the distant bead did not pass the pylorus (the bead was too thick to enter the narrow canal).

The above treatment being carried out for two weeks without any improvement of the ischochymia, and the patient having lost two pounds in weight, surgical intervention was clearly indicated.

On account of the constant presence of bile in the stomach, the diagnosis of a rigid pylorus, being stenosed and at the same time patent, was made.

On Jan. 12, 1908, nine P.M., the stomach was washed and no more solids or liquids were given. The next day at 1 P.M. the stomach tube brought up about 300 cc. of dark green contents containing gastric juice and bile. Food was not present; the acidity was 60, free HCl 40, no occult blood.

About 3 P.M. the patient was operated upon by Dr. Willy Meyer.

In the pylorus was found a thickening extending into the duodenum. The stomach was somewhat dilated with walls considerably hypertrophied. The gall bladder was normal. The liver looked blueish grey and had a somewhat uneven surface. A posterior gastroenterostomy was made. On the 14th and 15th the patient had gastric hemorrhages, which ceased. Later, pneumonia developed with very high temperatures and the patient finally succumbed on Jan. 19, 1908.

Epicrisis. Regarding the diagnosis of the above case it had to be made "stenosis and patency of the pylorus." The stenosis was certain, owing to the constant presence of ischochymia and occasional peristaltic restlessness. The rice test as well as the methylene-blue tallow bead also pointed to it.

The proof of the inability of the pylorus to close was not so easy and rested more on probability than absolute positive evidence. The constant presence of dark green bile in the stomach, whether food was taken or not, spoke for a continuous regurgitation of duodenal contents (bile) into the stomach.

This may be caused by two conditions: by a stenosis of the duodenum below the papilla Vateri, or through patency or inability to close on the part of the pylorus. In this case we had to deal with the latter condition. In the cases of stenosis below the papilla Vateri—which were first described by Riegel and two cases of which I have myself observed—bile is found in large quantities. The chyme is yellow, due to the presence of bile and contains a very small quantity of gastric juice; usually pronounced subacidity with absence of free HCl exists. In our case, however, the amount of bile was small, dark green in color, and the gastric juice was abundant with a high acidity. The assumption of a continuous partial regurgitation of small quantities of bile through the open pylorus appears therefore more plausible.

The operation showed that this was correct; there was no stenosis below the papilla Vateri, nothing abnormal was discovered in the gall bladder, and no dilatation of the duodenum existed which would have occurred if a stenosis of the duodenum at the papilla Vateri had been present. The stenosis as well as the rigidity of the pylorus were determined during the operation by means of palpation.

Some time ago I called attention to the occurrence of patency

and stenosis of the pylorus (stenosis et insufficientia pylori). In my paper, "A Further Contribution to Our Knowledge of Ischochymia,"* I said: "The constant or frequent occurrence of small quantities of bile in the stomach does not in my experience militate against the existence of a narrowing of the pylorus; on the other hand, it appears to me to point to a firm rigidity of this orifice, in consequence of which the latter is never completely closed. A thickened and rigid pylorus of this kind was found in the patient described above, Aaron L. and in Mrs. S., and in both cases a certain amount of bile was constantly in the stomach."

This affection I have observed in benign as well as malignant diseases of the pylorus. In as far as very little is found in literature on this topic, I thought it expedient to publish this case in detail. It resembles those published in 1897, and can well serve as a good example of stenosis and insufficiency of the pylorus.

* *Medical Record*, June 19, 1897.

PERITONITIS.

BY H. J. BOLDT, M.D.
Professor of Diseases of Women, New York Post-Graduate Medical School and Hospital.

In the consideration of this subject I shall deal with it entirely from the standpoint of a gynecologist.

In women, the main primary cause of peritonitis must be looked for in the genital tract, as becomes evident when we realize the frequency of infections during the puerperium: Gonorrheal infection; infections from local gynecological treatment, and infections from minor surgical interventions, the most prolific being curetting, which is rivalled, perhaps, by dilation of the cervical canal for sterility. In addition we have ovarian tumors; fibromata of the uterus, and carcinoma as factors in the etiology of peritonitis. The connection of the peritoneal cavity with the interior of the Fallopian tubes and the uterus must also be considered, because fluids injected into the uterine cavity sometimes find their way into the peritoneal cavity and may produce a rapidly fatal peritonitis. Only a few weeks ago a case of this kind came under our care in the Post-Graduate Hospital. A woman in a tenement house was curetted three weeks after her confinement, and an intra-uterine douche was given. She developed an intense and diffuse peritonitis, and her life was saved only by prompt surgical intervention, with subsequent appropriate treatment.

Clinically, it suffices to divide peritonitis into infectious and non-infectious peritonitis; from the etiological point of view, and from the bedside view, into acute and chronic peritonitis, either of which may be local or diffuse. The term " idiopathic peritonitis " is obsolete, because there is no such ailment.

The infectious forms of peritonitis may be divided into:

1. Septic peritonitis, which is caused by the *streptococcus pyogenes* and *staphylococcus pyogenes*. It may be caused also by *bacterium coli* and by the *pneumococcus*. The best examples of this variety of peritonitis are seen in puerperal peritonitis, and in the septic peritonitis following abdominal operations.
2. Tubercular peritonitis.
3. Gonorrheal peritonitis.
4. Peritonitis of mixed infection.

Non-infectious forms of peritonitis are caused by foreign bodies, under which heading we may consider intra-abdominal tumors with a twisted pedicle, such as ovarian tumors; tumors of the uterus; blood, as from tubal abortion or from a tubal rupture, etc.

Of all forms of peritonitis the puerperal streptococcic variety is the most dangerous to life. In this we should always look for an external cause. It is usually carried from the endometrium by the way of the lymphatics into the circulation. The course of bacteremia is frequently so rapid that the patients are dead before the pathological changes of peritonitis are completely established. No lymph exudate is found in these rapidly fatal cases, except occasionally in the pelvis. The intestines are not matted together; they are simply enormously distended and have lost their glistening appearance, which has given place to a dull red or reddish gray. Only a very small quantity of serous exudate is found in the peritoneal cavity, sometimes so little that it is not noticed at all. The patients who die of peritonitis and in whom much serous or seropurulent exudate and matting together of the intestines by adhesions are found on post-mortem examination, have had the disease more than two or three days, and they do not clinically belong to the class of *acute diffuse* puerperal septic peritonitis. When I make the statement that they do not clinically belong to the class of *foudroyant* septic peritonitis patients, it is because not a single case of that class can be saved by any known method of treatment. The streptococci are so virulent and the invasion of the system is so rapid, that the patients invariably succumb in from two to four days from the first manifestations of bacteremia; whereas in other cases a patient may occasionally be saved by timely surgical intervention.

Symptoms.—In instances of puerperal peritonitis, the disease is almost always ushered in by a more or less protracted and more or less violent chill, the character of which depends upon the virulence and the quantity of the poisonous microörganisms that have found access to the system. With this there is an increase in the pulse rate and an elevation of the temperature. The pulse-beats vary from 120 to 140 per minute and are frequently dicrotic. The temperature varies from 103° to 106° F. The temperature, however, should not be relied upon for a prog-

nosis, because very often it will drop to 100° F., or even lower; but the pulse does not harmonize with the drop in temperature and continues to be rapid and feeble. The lowering of the temperature is only temporary. Tympanitis is present and increases rapidly, so that the abdomen becomes ballooned. The abdomen is very sensitive to even the lightest palpatory touch, although when left alone the patients do not voluntarily complain of abdominal pain. The epigastric region imparts an elastic tensity to the touch; in this region the patients usually complain of a distressing, burning sensation. The facial expression is quite characteristic; it is pinched, especially about the nose. The color is often that of one slightly jaundiced. They eyes have a sunken appearance and lose their normal lustre. The tongue and lips are dry and parched, and the tongue is of a brownish color in the center. Intense thirst is a marked symptom as long as the patients are conscious; likewise frequent vomiting of brownish, bile-stained fluid. While obstinate constipation is present at first, later, perhaps only a few hours before death, profuse diarrhea may set in. Inability to empty the bladder spontaneously may be present very early, owing to paralysis of the bladder-wall from inflammation of its serous coverings. Hiccough, when present, is very obstinate and most distressing. Semi-consciousness and delirium are ominous symptoms of approaching death; the pulse is then frequently uncountable and almost imperceptible, and the rectal temperature very high, 106° F. or over. In these cases, while frequently we have been unable to show streptococci in the blood during life, we could always demonstrate their presence in the blood of the heart after death. Colon bacilli were also of very common occurrence on the peritoneal surface after death.

During the first few hours the diagnosis is not always easy, because a severe septic endometritis may give similar symptoms; but it is not long before the diagnosis becomes clear, the peritonitis making rapid progress and thus clearing up the case.

The uterus in all these cases is large and soft in consistency. There is no foul discharge from the genital tract; unless, as is not infrequently the case, there is a mixed infection, so that a sapric infection is associated with the streptococcic infection. The decomposition of the remnants left in the uterus then causes a discharge of putrid odor. It is on this point that, so far as my

observation goes, so many practitioners err in their diagnosis and prognosis. They are under the impression that, because there is no foul discharge, the case is not septic, and are thus prone to give a favorable prognosis. As a matter of fact, the cases of pure sapric infection, which always give rise to discharge of disagreeable odor, are not serious so far as the danger to life is concerned, so long as they are not complicated by a streptococcic infection. In instances of retained membranes the cervical canal is usually more patulous, so that the examining finger may readily be introduced through it.

There are cases of peritonitis in connection with the puerperium also of a septic character, that run what may be termed a chronic course, in contrast to the *foudroyant* type. The abdomen does not become so distended, nor is it so sensitive to palpation. Frequently any distension that has occurred recedes again, so that after ten days or more there is but slight meteorismus, and in some instances none at all. While the temperature may never exceed 102° F., and such temperature may be present only for a short time, perhaps for a few hours, and then drop again to 99° F.; and while the temperature usually ranges between 98½° and 100½° F., the pulse is not favorable, ranging from 110 beats and more to the minute, and being of small, compressible quality. The pelvic organs, especially the uterus, may have assumed their normal, or nearly normal condition, so far as the palpatory finding is concerned. Patients with this asthenic form of peritonitis may live two or three weeks or more, and then suddenly the temperature and pulse rate increase and the patients die. When the abdomen is opened after death, there is found in the peritoneal cavity, pus in greater or less quantity, but the intestines are not adherent to each other in any marked degree; they are simply bathed in pus. These cases, in my opinion, are always caused by a mixed infection. Pain is never a predominant symptom and may be absent throughout the illness. It will be found, however, that most of these patients have had one or more chills in the beginning of their illness, although these are not to be compared in severity with those occurring in *foudroyant* peritonitis.

For the very virulent type, that which is generally known as *foudroyant* puerperal peritonitis, I have no suggestion to make. My patients have always died, no matter what treatment was

employed. For those patients in whom, from the slower and somewhat milder cause of the disease, it is evident that the septic intoxication is not so virulent; in whom it is further evident that the streptococcic invasion proceeds from the uterus and that the parametria are not involved; in whom other appropriate treatment has not produced decided improvement in from twenty-four to forty-eight hours; and who are still in fair condition, I advise total extirpation of the pelvic organs. The operation must be done very rapidly, not more than from fifteen to twenty minutes being allowed for the entire operation. A slow operator, one who is not an expert in such work, should not attempt it.

This means, of course, that careful closing of the abdominal walls by tier sutures must be omitted; the flushing of the abdominal cavity should also be dispensed with, since it causes too much traumatism and deprives the intestines more or less of their endothelial covering, thus facilitating absorption. In short, the less one does in the opened abdominal cavity, the better are the chances for the patient's recovery. To use the words of my friend, Dr. Robert T. Morris, for abdominal work in general, "Get in and out again as rapidly as possible." A drain should be placed in the pelvis and carried into the vagina. The handling of the intestines should be avoided as much as possible. The patient should then have a narrow strip of gauze put over the abdominal wound, not the large bulky dressings usually applied to the abdomen after abdominal sections. Over the very simple dressing an oxide of zinc plaster binder of reliable quality should be placed, but not drawn so tightly as I have advised after abdominal sections for other causes. The patient should immediately be placed in a semi-sitting position; or, as I prefer, for a few days after such operation, the head of the bed should be elevated to an angle of from thirty-five to forty-five degrees, which can readily be done by the aid of the bed-elevator devised by me. The slipping-down of the patient toward the foot of the bed can be prevented by a large roll against which she is braced by her buttocks, and which is fastened from the sides to the head of the bed. Then an ice-coil is applied over the abdomen. A very ingenious ice-coil has been devised by Dr. A. Brothers. The constant instillation of saline solution, according to the method suggested by Dr. J. B. Murphy, of Chicago, should be begun. It has been

contended by some that the semi-sitting position is of no benefit; but experience has shown that peritonitis patients do better when it is employed, because secretions gravitate more readily toward the pelvis, and absorption into the system does not take place so readily from here as from the upper abdomen, close to the diaphragm.

I have saved a few patients ill with this disease by proceeding with the radical operation, done sufficiently early. One should not wait too long, however, and be deluded by a fall of half a degree or a whole degree of temperature. I have tried in a few instances to shorten the time of operation by putting on clamps as far out on the parametria as possible and cutting out the uterus close to the clamps, leaving them in the abdomen, relying on the hemostasis as we do in vaginal extirpation of the uterus with clamps. But in one case the patient had a fecal fistula following the operation, which required a long time to heal, and I have, therefore, abandoned this method, despite the fact that it shortens the time of the operation from five to ten minutes.

When the uterus is small, as after a criminal abortion, and such radical treatment seems indicated, the extirpation can be done through the vagina; but in full-sized puerperal uteri, the vaginal operation has not been satisfactory to me. I cannot do the work with sufficient rapidity; that is, within five minutes or less. It must be remembered that a prolonged anesthesia has a very detrimental effect on these patients. All antiseptic cleaning should be done before the anesthetizing is begun. Then only a little gas, followed by a few whiffs of ether or chloroform, will be required, especially if preliminary injections of scopolamine and morphine are made. The shock following operation will then be almost nil.

The general treatment, upon which a patient is put as soon as she comes under observation, consists of an intravenous infusion of saline solution, liberal alcoholic stimulation, ice-coil to the abdomen, and trunk elevation. While I have used the silver preparations quite frequently, and also anti-streptococcus serum, I have not seen any benefit from such therapeutic measures, although I still use them despite my former unsatisfactory results. I believe that in time a serum will be found which will be satisfactory.

In the still more chronic cases of peritonitis following delivery,

those in which none of the virulent symptoms are present, and in which the uterus does not seem to be a source of infection, I have practiced extensive vaginal section, with packing of the pelvic cavity with iodoform gauze. The results have varied, but have been mostly fatal. My experience with abdominal operations in such cases has been too limited to warrant me in expressing an opinion.

The wide opening of the cul-de-sac of Douglas, and loose packing of the true pelvis with iodoform gauze, as was originally advocated by Wm. M. Polk in the beginning of puerperal pelvic infection, is a most valuable prophylactic to diffuse peritonitis and serious inflammatory changes of the generative organs.

Patients with non-infectious peritonitis, as from the twisting of the pedicle of an ovarian tumor, of a pedunculated fibroid, or the rupture of an ovarian cyst, do not have the premonitory chill. Pain is the most prominent symptom in such cases. There may be some tympanitis, but it is rarely very marked. Thoracic respiration, if present at all, is not so marked as in puerperal peritonitis. The temperature is somewhat elevated and the pulse accelerated, but neither is much in excess of the normal. In these cases the pulse is never dicrotic, unless some complication sets in. Its quality usually remains good. In most instances non-infectious peritonitis remains localized. The severe systemic disturbances present in puerperal peritonitis are absent.

The treatment of non-infectious peritonitis is very simple. The cause must be removed. Such peritonitis rarely becomes diffuse. If it is due to a twisted pedicle of an ovarian tumor, or to any other recognizable cause, the abdomen should be opened and the tumor removed. In these cases neither drainage nor the semi-sitting position in bed is required.

Gonorrheal peritonitis may become diffuse, especially when developed during the puerperium. It does not differ in its symptoms from other forms of acute diffuse peritonitis, when the disease is at its height. It does differ, however, in the prognosis. Gonorrheal peritonitis is not as a rule a fatal disease; in fact, if it occurs outside the puerperal period, the prognosis is always good so far as life is concerned, if no serious complications develop, and if one does not resort to a surgical intervention, but treats it symptomatically on the expectant or conservative plan. Even if the disease is developed during the puerperium, the

prognosis is by no means so unfavorable as that of a puerperal septic peritonitis. The majority of cases recover. The positive diagnosis depends upon the finding of gonococci in the genital tract.

While diffuse peritonitis of gonorrheal origin during the puerperium is not nearly so dangerous as streptococcic peritonitis, or peritonitis caused by a mixed infection, it is not of such absolutely good prognosis as some authors would have us believe. While perhaps the greater number of patients will recover without surgical intervention, there are doubtless some cases that would be fatal if not operated upon. Leopold has published (*Centralblatt für Gynäkologie*, 1907) a very interesting instance of exceedingly virulent diffuse gonococcic peritonitis, from which, according to the description, the patient would undoubtedly have died, had he not made the abdominal incisions and instituted drainage. In such instances an extensive vaginal section, with the placing of a drain in the cul-de-sac, should be added. If a patient's condition is not improved after two days' treatment, it is better to resort to operation. I have but very limited experience with diffuse puerperal peritonitis of gonorrheal origin, but it seems to me that an extensive vaginal section with drainage, the semi-sitting position, the application of an ice-coil, and the internal administration of opium to the extent advised by the late Dr. Alonzo Clark, suffice to bring about a favorable result. While opium as a rule should be deprecated in peritonitis, I am confident there are exceptions. It is likely that it was in just such cases of puerperal peritonitis that Clark had such good results, even without surgical intervention.

Tubercular peritonitis is usually insidious in its onset. In most instances it starts from the pelvic organs, though the starting point may sometimes be from another part within the abdomen, as the intestines. In such cases the interior of the tubes is rarely infected with tuberculous products and the tubercles are then usually limited to the serous covering of the tubes. Clinically, it may be divided into the dry and the exudative varieties.

In the exudative variety more or less fluid is always found in the peritoneal cavity. The fluid may be diffuse or it may be encapsulated, forming sacculated tumors in various parts of the

abdominal cavity, and not infrequently causing an erroneous diagnosis. The fluid may vary from clear ascitic fluid to a purulent exudate. The walls of such exudates are made up of intestines which have become adherent to each other, together with the mesentery and the omentum, or of the intestines alone.

In the adhesive or dry form, the abdominal contents are matted together to a greater or less extent. Small tubercles and tubercular caseous nodules are frequently met with. The free serous ascitic fluid of tubercular peritonitis does not differ in its objective symptoms from ascites due to other causes.

If the tubercular peritonitis is caused by ascension from the genital tract, the infection may have come from inoculation from external sources; as from intercourse with one affected with tuberculosis of the testicles, or from a finger contaminated with sputum containing tubercle bacilli. We may also have tubercle bacilli conveyed by means of the blood-current from pulmonary tuberculosis or tubercular processes in other parts of the body. Whether primary tubercular peritonitis occurs, is a mooted question. It seems probable that the supposed instances of primary tubercular peritonitis had their origin in a tubercular intestinal ulcer. Because of its insidious character, most patients with tubercular peritonitis have had the disease for some time, before a physician is consulted. Sometimes the primary tubercular process has practically healed before we see the fully developed peritonitis. While the temperature of such patients may not be high, varying from 99° F. to 101° F., I have never seen an instance where for twenty-four hours it was entirely normal. Pain is never a marked symptom, though usually some pain is complained of upon palpation. The patients complain of a general weak feeling, and are more or less emaciated. If the disease started from the pelvic organs, the adnexa can always be palpated. They are thickened and indurated. In the cul-de-sac of Douglas an exudate may often be felt, which is moderately painful on pressure with the examining finger. Very frequently tubercular nodules may be felt per vaginam, or per rectum. The ovaries are sometimes changed into tubercular tumors two inches or more in diameter, and upon examination give the impression of being dermoid tumors. The diagnosis in many cases is by no means easy, because of the varied subjective and objective symptoms presented by different patients. The subcutaneous injec-

tion of tuberculin has several times aided me in making a diagnosis. In all suspected cases we should look for tubercular lesions in other parts of the body, and if these are found it will aid us much in coming to a conclusion. It is sometimes particularly difficult to differentiate a tubercular peritonitis with free ascites from peritonitis caused by carcinoma, because in the latter also we have free ascitic fluid and the formation of small nodules. The condition is probably carcinomatous if we can palpate a *large* tumor of the ovary, which is diffusely adherent. Nodular formations in the retro-uterine space are of more frequent occurrence in patients with carcinoma than in those with tubercular disease.

While a spontaneous cure of tubercular peritonitis is not impossible, such cure is very rare, and we must look upon the disease as being surgical, especially if the primary lesion is in the genital tract. Abdominal section with extirpation of tubercular Fallopian tubes, without attention to the uterus also, is likely to mean only a prolongation of life. Such patients should have also a very thorough curetting with energetic local application of carbolic acid to the endometrium, or, what I consider a still safer procedure for the patients' future, a complete extirpation of the uterus. Twice it has been necessary for me to extirpate a uterus per vaginam for tubercular endometritis, subsequent to an abdominal operation for tubercular peritonitis with salpingitis. This was in my early career, before I operated so radically as I do now. In other instances during that period, where I did only an abdominal operation, some of the patients subsequently died of tuberculosis, in periods varying from three to six years after the operation.

Good hygiene and nutritious diet are most important adjuvants in the treatment.

From our present knowledge and the experience that has been gained with surgical intervention, the disease should be looked upon from a surgical standpoint, because the direct mortality from abdominal section is very low and much benefit may be derived. In fact, if the disease starts in the genital tract, and has not advanced too far, the greater number of patients are cured if the operation is sufficiently radical. I have always employed drainage in the exudative variety of tubercular peritonitis for a few days subsequent to operation,

but never in the adhesive or dry form. The latter form gives the more unfavorable prognosis. The employment of X-rays seems to be beneficial in this form of peritonitis.

The most frequent peritoneal inflammation that a gynecologist comes in contact with is that form limited to the pelvic peritoneum, which, in contradistinction to the other varieties of peritonitis, is called pelveo-peritonitis. Here, too, a division into acute and chronic pelveo-peritonitis suffices for clinical purposes. It is especially important for us to recognize these localized peritoneal inflammations, because with the employment of improper therapeutic measures they are liable to be transformed into diffuse peritonitis.

As a rule, pelveo-peritonitis has its beginning from an infection of the pelvic organs, gonorrheal infection playing a most important part in its production. A gonorrheal salpingitis leads in most instances to a more or less extensive inflammation of the pelvic peritoneum and an agglutination of the Fallopian tubes with the adjacent pelvic peritoneum. The tubes become adherent to the ovaries, uterus, intestines, and omentum. The cause of the more intense pelvic inflammation is the escape of some infectious secretion from the fimbriated extremities of the tubes into the pelvic cavity. While in the beginning the adhesions are not firm, in later stages they may become so firm as to require considerable force to separate them. We may have as the result of such inflammation only an agglutination of the pelvic organs with each other, or also with the structures lying in the pelvis; or we may have the production of an exudate in the pelvis. Such exudate may be serous, sero-purulent or purely purulent, then forming a pelvic abscess.

In the exudative form of pelveo-peritonitis, in which streptococcus or staphylococcus infection is usually present, the formation of a retro-uterine tumor may be prevented if there has existed a previous chronic pelvic inflammation which has caused the cul-de-sac of Douglas to become wholly or partially obliterated. If the exudate is large, the uterus is pushed upward, and the vaginal portion of the cervix will be found near the symphysis. The posterior vaginal wall then bulges downward, its base in some instances reaching almost to the vulva. The pelvic pain in instances of a large exudate is sometimes excruciating. Rectal tenemus is a marked symptom, as is also the passing of mucus of a glairy

character *per anum*. Such exudate always imparts to the examining finger an elastic feeling, varying in degree with the tensity of its walls. Fluctuation, more or less marked, is invariably present. In two cases I have seen the exudate anterior to the uterus. When the cul-de-sac is obliterated entirely, and the exudate is higher up in the pelvis, the diagnosis becomes very difficult. We can feel the exudate, but cannot always be sure of its nature, because of the uncertain feeling it imparts to the examining finger. The subjective symptoms do not help us to determine the character of the exudate. In the serous, the sero-purulent, and the purely purulent exudate, a slight elevation of temperature may be present. A purulent exudate, as a rule, feels a little doughy and the vaginal mucous membrane imparts a more succulent feeling than in the case of a serous exudate.

Only a few days ago I made a mistake in a case which was so instructive from another point of view, that I quote it. The woman had had an abdominal section done at other hands; I thought it to be a supravaginal hysterectomy for what I had reason to believe was a small myoma. Sometime after the operation she presented herself to me, complaining of pelvic pain, rectal tenemus, and obstinate constipation. Examination revealed an exudate in the pelvis, directly behind the cervix. It was smooth at its base and globular in contour. I believed it to be a retro-uterine hematoma following from some oozing after the operation. About two weeks later I again saw the patient, who then complained of an increase in intensity of the previous symptoms. Examination revealed that the exudate had increased very much in size and was distinctly fluctuating, and that its base came almost to the vulva. I diagnosticated that the hematoma had become changed into a pelvic abscess. On the next day I made a vaginal section. About 200 c.c. of serum escaped, and on examining the pelvis I found that above the exudate was a hard, indurated mass, which made the impression of a malignant neoplasm. Because of the universally fluctuating sensation on examination, I could make no exact diagnosis without knowing that malignant disease was present.

While a leucocytosis of 20,000 speaks in favor of pus, this sometimes occurs in instances of serous exudate. A large retro-uterine serous exudate of pelveo-peritonitis may be mistaken for

an ovarian cyst which has become adherent to the floor of the pelvis, in Douglas's cul-de-sac. In the latter instance we can, as a rule, map out the boundaries of the tumor; whereas in the case of an exudate these boundaries consist of the intestines, uterus, and pelvic wall, and cannot be so clearly defined as in an ovarian cyst.

To differentiate a serous exudate from a fresh retro-uterine hematocele, from the objective symptoms, without taking into consideration the history, is, in my opinion, impossible. But this is not difficult, when the blood has begun to coagulate, so that in some parts fluid is felt, and in other parts harder masses, especially if we can elicit the history of a probable tubal abortion.

There is a difference of opinion as to the treatment of retro-uterine exudates, so long as they have not undergone purulent changes. Some surgeons treat them on the expectant plan, with an idea that they will spontaneously become absorbed. While I have treated some cases by this method, and in a few instances with success, I do not believe it to be a correct procedure. My practice is always to make a vaginal section, if permission is given me to operate. This course is quicker and more in the interest of the patient. For that reason I do not sanction the use of an exploratory puncture with a needle, unless I purpose to follow it immediately with a vaginal section, and then to pack the cavity loosely with gauze. The section should always be made extensive, large enough to permit the operator to put his hand into the pelvis and palpate its boundaries, so as to see if there are any unopened pockets. This is best accomplished with the pelvic abscess instruments devised by me for such purpose. No matter what the character of the retro-uterine exudate is, my treatment is always of that kind, except in some instances of hematocele.

Other cases of acute pelveo-peritonitis should be treated by the expectant plan, absolute rest in bed, the application of ice-bags to the lower abdomen, and light diet. If the acute stage has passed off, circumstances must guide us in the further treatment.

In vesical tenemus, so commonly present in acute pelveo-peritonitis, relief may sometimes be obtained from the administration of the homeopathic mother tincture of bryonia in six to ten drop doses, given at intervals of six or eight hours.

In some instances of chronic pelveo-peritonitis the diagnosis must depend upon the palpatory sensitiveness of the plevic peritoneum. When the inflammatory process is circumscribed and limited to small areas, as in the case of neurasthenic patients, it is sometimes quite difficult to determine whether the pain caused on palpation is due to the patient's neurotic condition, or to a low grade of inflammation. This can generally be determined by palpating different parts of the pelvic peritoneum, and ascertaining whether pain is complained of alike in all parts, or is localized at or about the same points. After an acute attack of pelvic peritonitis has passed off, it will sometimes be found that various dislocations of the pelvic organs have taken place.

Although it has not been possible, in so brief an article, to go into the details of all phases of peritonitis from the stand-point of a gynecologist, yet the more important points have been considered.

OPERATIONS FOR THE CORRECTION OF DEFORMITIES FOLLOWING POTT'S FRACTURE.

BY WARREN O. PLIMPTON, M.D.
Professor of Orthopedic Surgery at the New York Post-Graduate Medical School.

Deformities following Pott's fracture, are not infrequent. The disability, discomfort and pain attending such conditions are usually in proportion to the degree of deformity. These deformities are serious and furnish a decided impediment to the leg in bearing the weight of the body. The effect of extreme deformities is to render the foot almost useless. A patient once said to me that he would prefer amputation and an artificial foot, if the trouble could not be remedied by an operation. Braces, and other forms of support have been devised as an aid in walking, but they are in no wise curative; are unsatisfactory in application, and must be worn constantly. Operative treatment alone, offers the only means for permanent relief.

Before discussing the different operations for correction of these deformities, I wish briefly to review the anatomical conditions existing with this fracture. The typical Pott's fracture occurs about three inches above the tip of the external malleolus, produced by forcible eversion and abduction of the foot. (As seen in diagram B.) . Two other fractures often occur at the same time: one, of the outer lower edge of the tibia (see Fig. A); the other, of the inner malleolus, as in Fig. A. The inferior tibio-fibular ligament at point E in Fig. B. sometimes yields instead of the edge of the tibia, and the internal lateral or deltoid ligament, may be torn without a fracture of the inner malleolus (see Fig. B). Pott's fracture may be produced. also by forcible inversion of the foot, (see Fig. D). The fibula is broken from one, to one and a half inches above its tip. The inner malleolus may be broken obliquely upward and inward also, (see point F, Fig. D) as in forcible eversion of the foot. The so-called mortise in Pott's fracture is so much widened and enlarged that it allows the astragalus to become displaced both outward and backward. The inner lower edge of the tibia is prominent; a depression appears over the break in the fibula; the foot is pushed backward, everted and turned

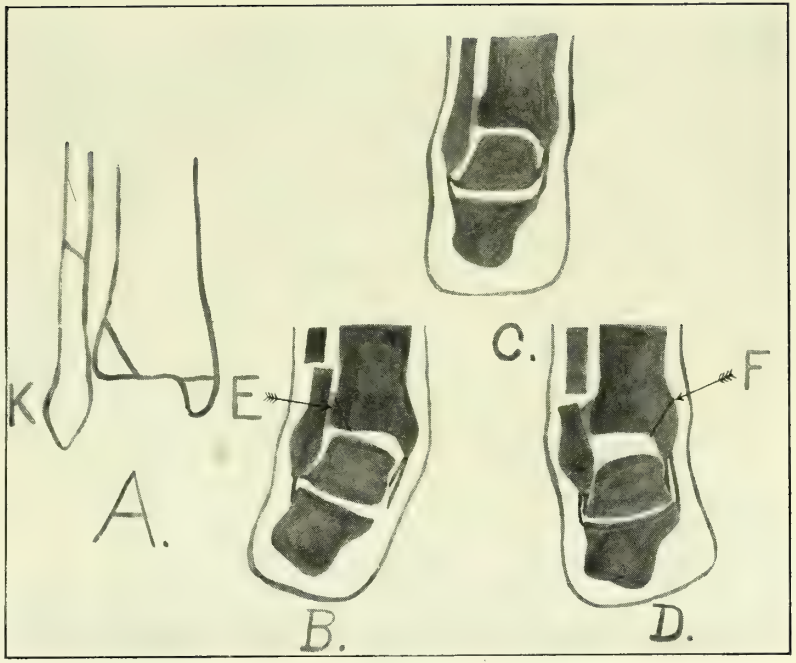


Diagram showing a perpendicular section through the ankle joint, with position of the bones after Pott's fracture and the sites of fracture.

(Plimpton)

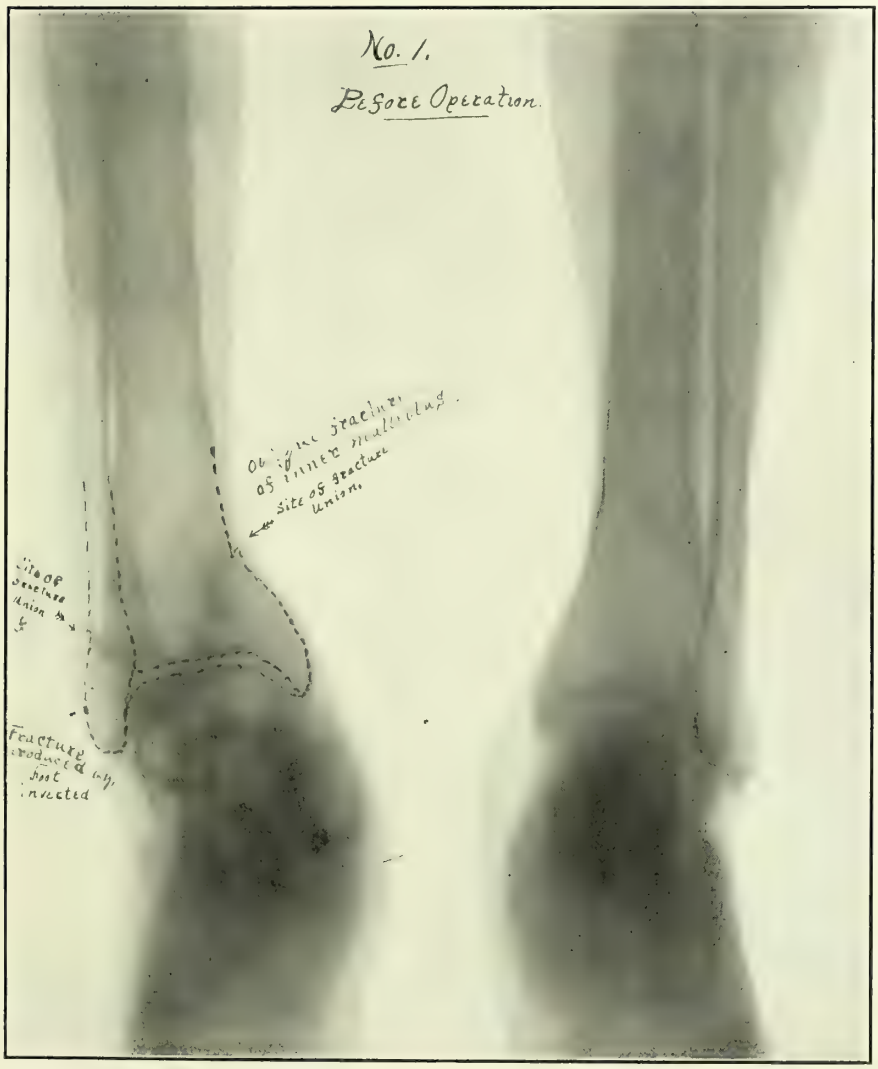


FIG. 1.—Shows bad position of the bones, united after oblique fractures of the inner malleolus and fibula. Fracture, produced by too forcible inversion of the foot.

(Plimpton)

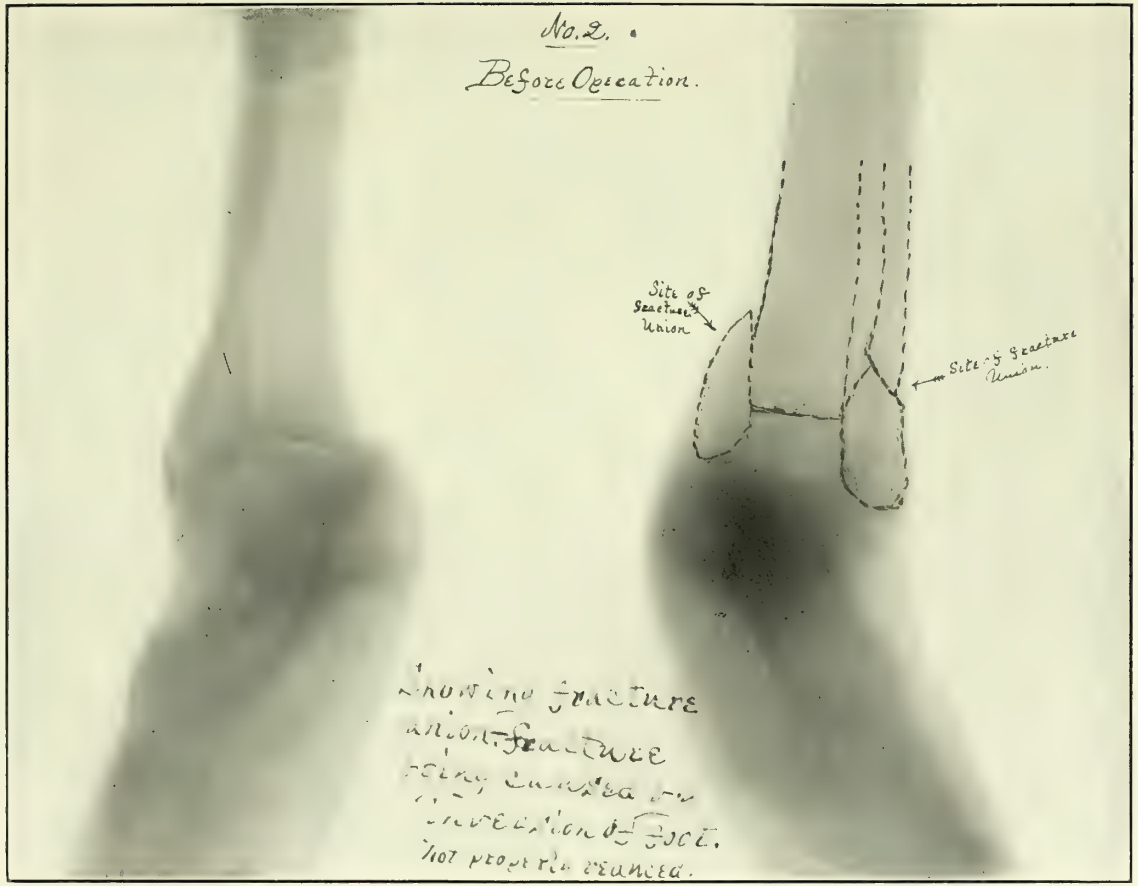


FIG. 2.—Showing line of fracture still unreduced. Fracture caused by too forcible inversion of the foot.
(Plimpton)

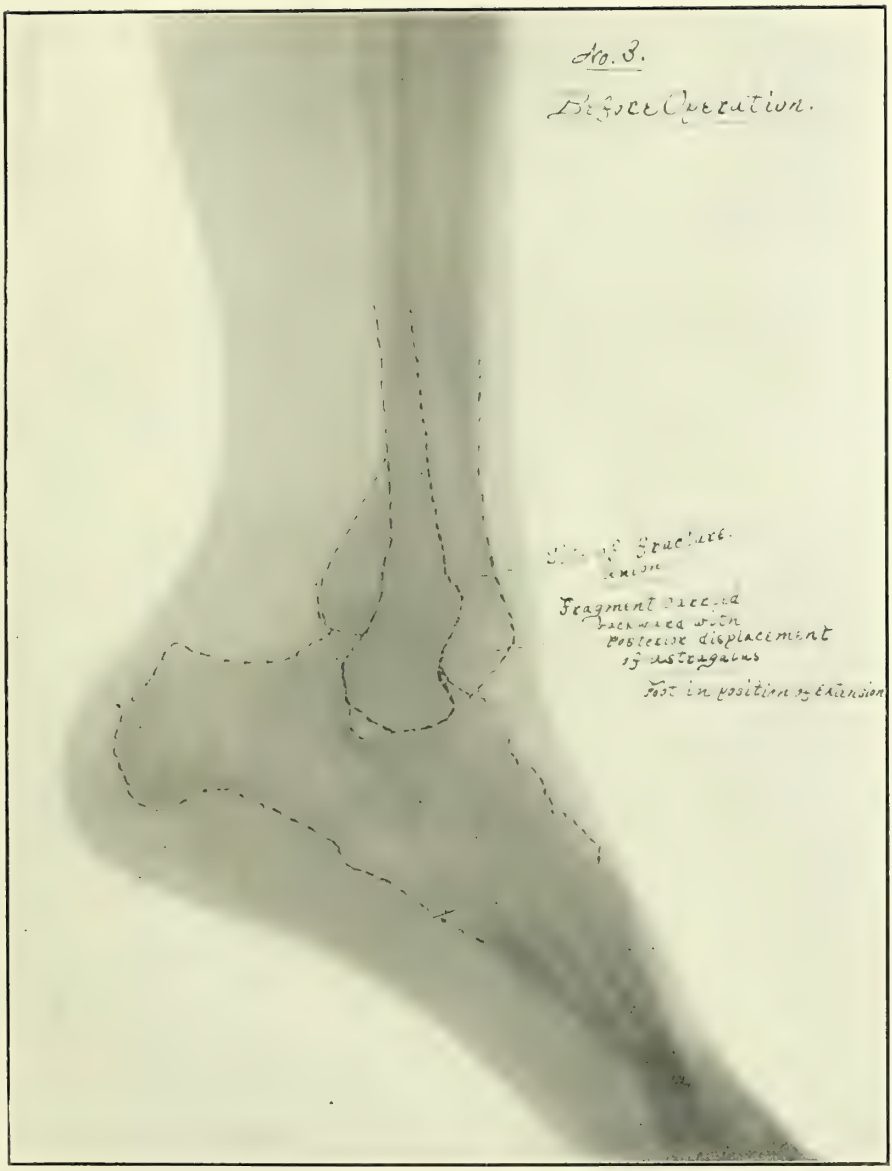


FIG. 3.—Shows position of the bones with union before operation. Fragment of fibula carried backward, with posterior displacement of the astragalus and foot in position of extension. (Plimpton)

out. These are the deformities seen in vicious union, except in certain cases of over-correction where inversion takes the place of eversion, as in Fig. D.

The degree of dislocation, whether partial or complete, together with the exact relative position of the bones, determine the extent and nature of the operative measure necessary to overcome the deformity. It is for this reason that no one operation can apply to all cases, although a definite method or mode of procedure may be followed to some extent. The radiograph has, within the last number of years, been of invaluable service in studying these cases, by revealing the exact position of the bones. By comparing a series of radiographs, the variety of conditions noted is striking. The amount of lateral displacement and eversion of the astragalus may vary to a considerable extent. In one, the backward displacement is greater than in another, since the tibia and fibula are more widely separated in some cases than in others. The foot, in backward dislocation, is extended and the fragments are driven backward as seen in radiograph No. 3. In case of fractures of the outer edge of the tibia, the outer upper edge of the astragalus is driven up between the two bones of the leg. We find eversion marked in these cases and the foot greatly out of line with the axis of the tibia. Besides the everted positions of the foot in deformities following this fracture, there may occur also an inverted displacement, the result of over correction or the violent inversion of the foot when the fracture is produced. An oblique fracture of the fibula and inner malleolus (see Fig. D) makes this possible, especially when the type of the break is not recognized and the deformity goes unreduced.

I have two radiographs, Nos. 1 and 2, given to me by the late Dr. A. M. Phelps, showing this condition of inversion. The inner fragment is pushed upward on the tibia, while the fragment of fibula is drawn downward. (very marked in radiograph No. 1). I call attention to these anatomical differences especially to emphasize the fact already stated, that no one operation can apply to all. The time elapsed since the injury regulates, to a great extent, the ease with which the foot can be restored to its normal axis. An operation within two or three months after the fracture would more easily overcome the deformity, than if attempted later, when the tendons, ligaments,

fibrous tissue and muscle have undergone the change of becoming shortened in some places and lengthened in others. Were the original fractures to be reproduced in a deformity of long standing, the change in the surrounding tissues would prevent its reduction. Not only do soft parts change and make later operations difficult, but bone surfaces become altered in contour from new points of pressure and by the constant motion and use of the part.

The position of the foot, the severity of the injury, the age of the patient, the time elapsed since the fracture, and the amount of work that has been imposed upon the ankle, all have an important bearing on the operative measures suited to each case. While the X-ray shows non-union and malposition, it does not tell whether or no the adjacent soft structures will yield after the bones have been operated on. This must be determined by trial, especially in extreme conditions. The quite extensive cutting of soft parts is often necessitated before reduction can be accomplished. On account of these difficulties and the possibility of destroying the mobility of the ankle, operations, external to the point, either adjacent to or farther removed, have been devised and recommended; their purpose being to restore the foot to the axis of the leg, so that the patient's weight is borne on the outside of the foot. For example, the operation of cuneiform osteotomy aims to do this by removing a wedge of bone from the tibia, two or more inches above the lower articular end, with a fracture or osteotomy of the fibula at a point opposite. This allows the foot to swing into line with the axis of the leg. Some operators employing this method have reported useful feet and satisfactory results. Linear osteotomy through both bones at the same location, has accomplished a similar result. In neither of these operations is anything accomplished by way of overcoming the dislocation or malposition of the bones which resulted at the time of injury, an important element in producing a useful joint. The horizontal tibial articulation also is changed to an oblique position. In fact, it is the production of one deformity to help out another. Hence, I believe, operations that aim to restore the broken and displaced bones to their original relations—operations that deal directly with the injury—offer a much better chance for a useful foot and ankle, than those that seek only to

alter the perpendicular line of the leg, so that it falls through the center of a series of deformities to the outer weight bearing part of the foot.

An operative method of overcoming deformities following Pott's fracture, should have for its accomplishment a restoration of the foot to the perpendicular axis of the leg. The superior surface of the astragalus should rest squarely on the horizontal plane of the tibia. The mortise of the joint should be as perfectly restored as possible, although this cannot always be accomplished, especially in extreme cases; in these, resections are necessary. If we could reproduce the conditions existing directly after the injury and then treat it as a recent fracture, the problem would be solved. This plan, however, will do for recent cases, as there is usually little change in ligamentous and fibrous tissue. This same procedure of operation was set forth by Dr. Irving S. Haynes, of New York, in a paper read October 7, 1891, in which he says: "Restore the limb to the condition it was in at the time of the injury, and then treat it as a recent fracture." In this paper he reports a case of a man 38 years, upon whom he operated 3½ months after the injury, by osteotomy through the fibula and inner malleolus, at the site of fracture. He then was able to overcome the everted position of the foot and reduce the dislocation. The subsequent treatment was the same as after immediate injury, and the result a straight limb, movable joint, and strong ankle. Dr. Haynes reports the same operation on another patient, with deformity of a few weeks standing, with good result. He also suggests a plan to reduce the increased diameter of the ankle, (in certain cases where new tissue fills the gap or space between the bones of the leg); by going in between the tibia and fragment of fibula and removing any new tissue that may have formed. The bones then can be approximated in a way to restore a normal mortise. I had occasion to remove ½ inch of new bone from between the fibula and tibia, while operating upon a deformity of this kind, which I will mention later.

By way of further illustration of suiting the operation to the condition found, I refer to two cases of deformity following a recent Pott's fracture, one in a boy of sixteen years of age, and the other in a woman of middle life, which were operated upon by the late Dr. George F. Shrady. He reproduced the original

fractures by osteotomies, and was then able to overcome the deformity. The subsequent treatment was the same as in a primary fracture. The results in both cases were good. The radiographs numbered 1 and 2, are from patients of the late Dr. A. M. Phelps, whom I assisted in operating for the correction of these deformities. These show an inverted position of the foot, due to over-correction. The fragment of the inner malleolus is seen displaced upward on the tibia, while to the outer side of the leg the fragment of the fibula is drawn downward, holding the foot in the position of inversion, causing a consequent disability in the use of the limb. No. 1 shows the foot of a woman between 50 and 60 years of age. Number 2 is that of a man about 20. Both were operated on about one year after the fracture occurred. The operation employed was a division of the bones with a chisel at the point of original fracture. Both feet could then be placed in the normal position. They were dressed with Plaster of Paris over the usual soft dressings. The foot was placed in a straight position with the axis of the leg, care being taken to flex the foot slightly. The result in both cases was good—perfect position and good motion.

In the above six cases, the same operative procedure has been followed by good union of parts, with satisfactory results. No wiring of fragments seems to have been necessary to keep the bones in apposition. The wire, however, might be used in any case requiring support to hold the fragments in apposition. If, in this plan of operation, after a division of the bones originally fractured, it was found that the soft parts resisted, tenotomies or division of ligaments might sufficiently free the foot, so as to make reduction easy. This having failed, it might be necessary to resect the bone that interfered with the reduction, or, if the surrounding soft tissues were still at fault, together with the bones, it might be necessary to do a complete excision, thereby getting ancyhlosis; but even so radical an operation gives a useful limb. The foot can then be placed in the proper axis of the leg. If slightly flexed and inverted a trifle, so as to allow the weight of the body to come on the outer side of the foot, the patient can walk well.

Dr. Stimson, in his book on fractures, tells us that excision of the ankle has been frequently resorted to by the French surgeons with satisfactory results. Dr. Phelps found excision

necessary in a case operated on by him in 1888. He reports anchylosis with a useful limb. Complete excision is a last resort for very extreme cases, usually of long standing, where nothing short of it will overcome the conditions found. In most cases of extreme deformity following Pott's fracture, if the deflection of the foot cannot be overcome because of shortened tissue or changed articular surfaces, resection of the bone making up one side of the articulation, preferably the tibia, will in most instances make the foot movable enough to bring it in line with the axis of the leg. If the resection is confined to one side of the joint, without disturbing the cartilage on the other, a movable joint, as a rule, results. Again, suit the procedure to the requirements of conditions found. As in the case of the deformity shown in radiograph No. 3, where the astragalus is displaced backward, carrying with it the lower fragment of the fibula, which has united in that position. The foot is extended. Dr. Phelps, who was the operator, reported that after dividing the fibula, he was unable to flex the foot or bring the astragalus into a correct position beneath the tibia. Entering the joint from the inner side, he resected enough, from the upper surface of the astragalus to allow the foot to come forward. The result was a movable ankle and useful foot.

April 1, 1893, Dr. Shrady published in the *Medical Record*, a report of an operation devised and performed by him, to overcome extreme outward and backward dislocation of the foot following Pott's fracture of two year's standing, with ununited fragments of fibula. As far as I know, this may be called Shrady's operation, and is especially adapted to such extreme cases as he mentions. This, too, like the other operations for the recent conditions, must be modified to suit the case. In describing it, I quote from his article. He says: " The amount of surrounding consolidation was so great, the axis of deflection so extreme, that I concluded to devise a method that would apply to this particular case. I resected the lower portion of the tibia, and also the lower and ununited fragment of the fibula. The fragment of the internal malleolus was found displaced downward and forward. The bone was sawn through in situ, in the usual manner, with the least possible injury to the soft parts. This was in great part due to the use of the subcutaneous saw. The periosteum was preserved and its deeper

attachments carefully dissected. The astragalus was not disturbed. The tendo-Achillis was divided; the sawn plain surface of the tibia was placed in contact with the articular surface of the astragalus, thorough drainage of the joint established, aseptic dressings applied, and the foot and ankle were enveloped in a plaster splint including the knee-joint and thigh. At the end of five months the new joint was formed. Nine months after the operation he had good motion of his joint, and was able to walk upon it without assistance of cane or brace."

In May, 1896, a gentleman, 35 years of age and weighing 225 pounds, came to my office on crutches, to consult me about his foot. Eight months before, he had stepped into the turntable pit in a railroad yard, falling a distance of twelve feet, striking on his feet, producing a Pott's fracture of the right leg. He had been treated by two physicians in a neighboring city, but up to the time I saw him had been unable to walk without the aid of crutches. The foot and ankle presented the characteristic appearance of an outward deformity following Pott's fracture, with much eversion and outward displacement of the foot, as shown in a photograph No. 4 taken at that time together with a radiograph, No. 5.

It shows exactly the points of fracture and the displacement of bones. The fibula has united with its lower end deflected outward; the inner malleolus is still separated from the tibia, but united by fibrous union. The bones of the leg are separated, and a growth of new bone appears on the inner side of the fibula. The upper, outer corner of the astragalus is resting between tibia and fibula. On June 15, 1896, I operated on the ankle, at the New York Post-Graduate Hospital, following in principle the method of reproducing the conditions existing immediately after fracture. After first making the Langenbeck incision for excision of the ankle, I divided the fibula with a chisel where it had been fractured. (See Radiograph No. 5.) A curved incision around the end of the tibia through the fibrous union of the inner malleolus, opened the joint. An attempt to reduce the dislocation was then made without success. With a chisel, $\frac{3}{8}$ of an inch was then resected from the horizontal plane of the tibia, (see radiograph No. 5) and the cut surface so gauged out as to conform to the upper surface of the astragalus. The foot could now be brought only partly into line—the growth

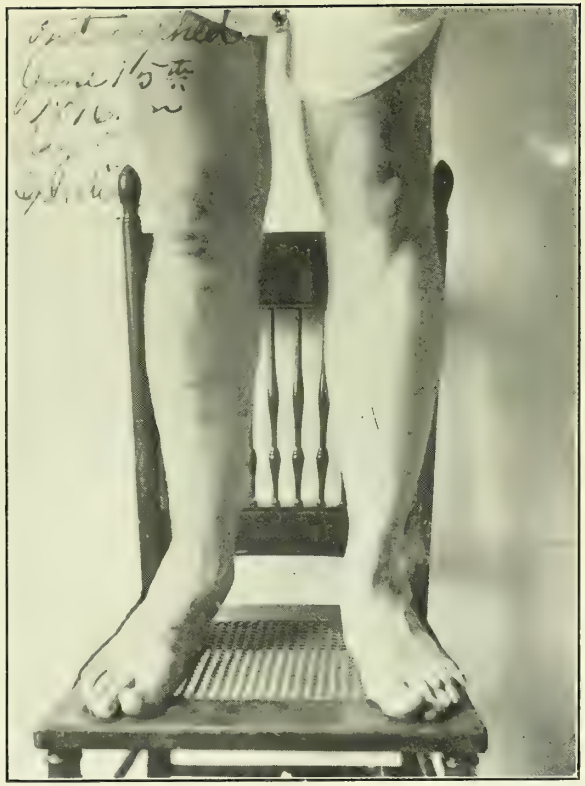


FIG. 4.—Shows eversion of the right foot on June 15, 1896, on day of operation.
(Plimpton)

FIG. 6.—Shows position of the right foot 4 years and 7 months after operation.

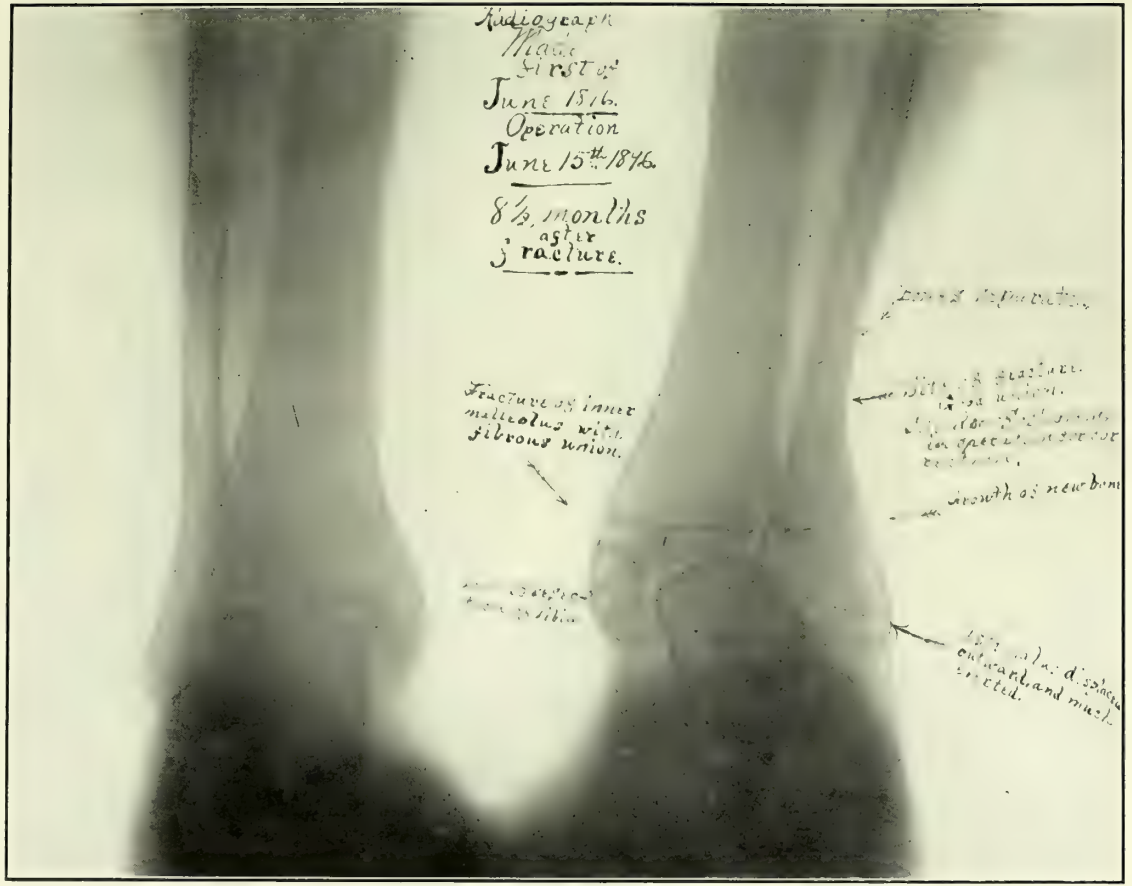


FIG. 5.—Radiograph made in June, 1896, 8½ months after the accident and just before operation. Shows fracture of inner malleolus, eversion of astragalus, site of fracture of the fibula and growth of new bone between the tibia and fibula. (Plimpton)

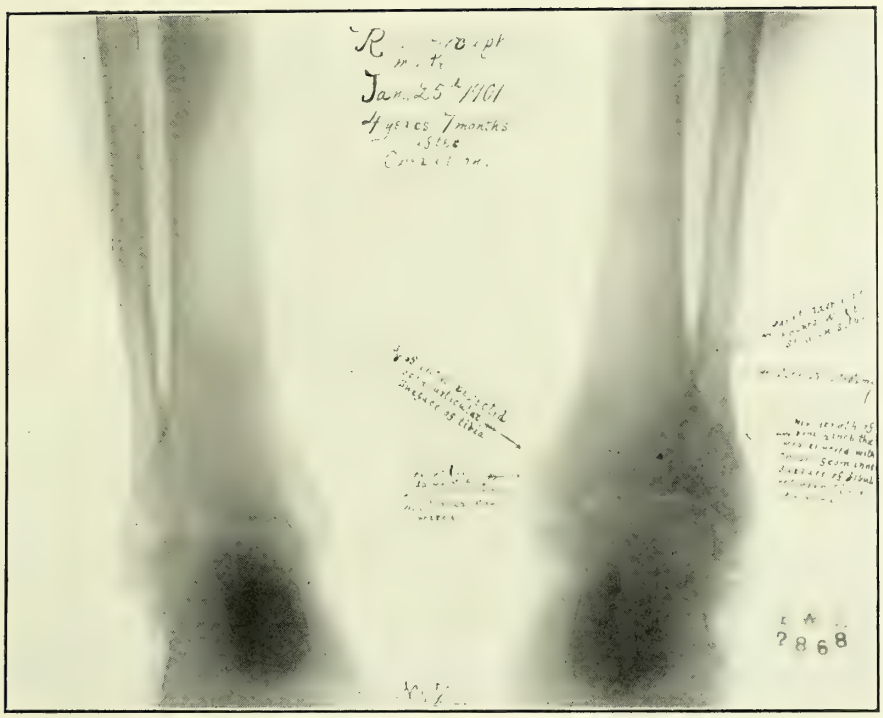
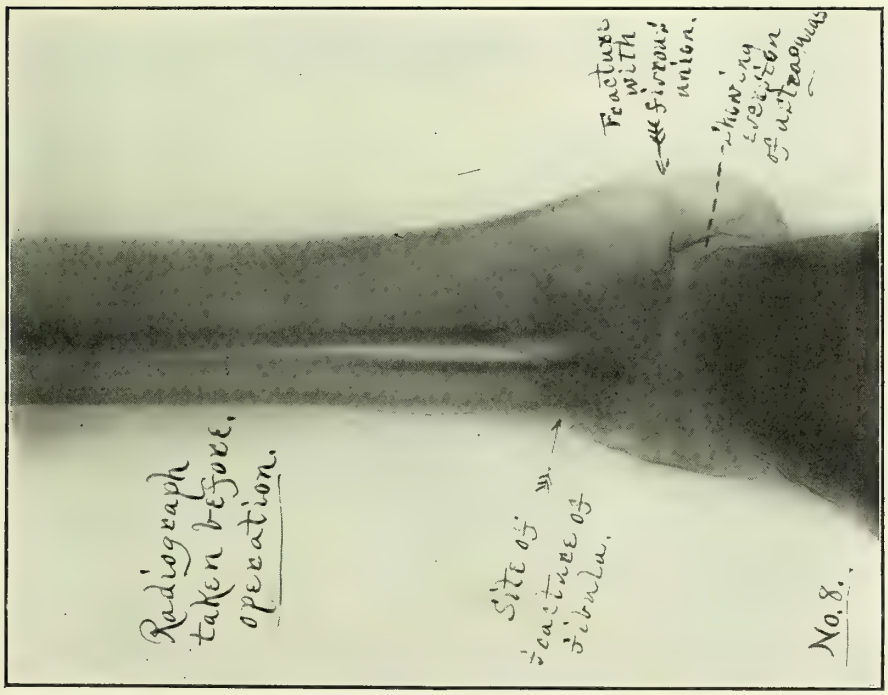


FIG. 7.—Shows result of operation of Fig. 5, 4 years and 7 months after. Good union of parts, good position of bones, and useful joint. Iron wire still in place.



(Plimpton)

FIG. 8.—Before operation. Antero-posterior view, showing fracture of the inner malleolus, eversion of the astragalus and fracture of the fibula.

of new bone between tibia and fibula prevented. With a chisel through the outer incision, I removed this growth of bone, which was ½ inch thick. The foot could now be placed under the tibia and a good mortise formed for its support. The fragment of fibula was then wired with iron wire. Fibrous tissue was removed from the base of the inner malleolus, which was then wired to the tibia with two loops of iron wire. The tendo-Achilles was next divided so as to allow absolute control of the foot. Catgut drainage was used, the usual dressings applied inside, with a Plaster of Paris dressing over all, extending above the knee. The foot was very slightly inverted and flexed. Union went on uninterruptedly. The external incision closed entirely in three weeks, and the patient left the hospital in six weeks from date of operation. He returned to his business September 7, still using the crutches and wearing the plaster below the knee. On November 1, 4½ months from the date of operation, the plaster and crutches were discarded, and he has walked on the foot ever since. He walks without a limp, has lateral motion, and a range of flexion and extension (through an arc of 30°) at the ankle sufficient to walk gracefully. January 25, 1901 another radiograph, No. 7, was made, which shows the joint 4½ years after the operation. The bones are somewhat thicker than in the other leg, but still solid and in good position. Photograph No. 6, taken at the same time as the radiograph, shows the external appearance of the ankle, somewhat thicker than its fellow, but nevertheless strong and in good position. I would call attention to radiograph No. 7 which shows the faint dark lines of the iron sutures, still in situ 4½ years after operation.

In May, 1907, Dr. M. aged 35 years, of Fort Worth, Texas, consulted me about a deformity of his left ankle, following a compound Pott's fracture received October 25, 1906, about 7 months before. He was using crutches and could bear little weight on the left foot. Radiographs Nos. 8 and 9 show the position and the condition of the bones at the time I first saw him. He expressed himself as follows: "The foot is worthless as it is now; do what you think best; I have nothing to loose, but everything to gain." Accordingly, on May 16, 1907, I operated. I followed, in a general way, the operation described in the previous case. I exposed the site of the fracture of the fibula and found that there had been an oblique fracture, which had united in the

usual deformed position, with lower end of the fibula thrown obliquely outward, with the ends of fragments at the site of fracture drawn in towards tibia. With my chisel I divided the fibula obliquely as nearly as possible in the line of the original fracture. The tip of the internal malleolus had been torn off and had united by fibrous union, as in the previous case. I made an incision through this fibrous union and removed the fibrous tissue from the approximate sides of fragments. This done, I was unable even with great force to restore the foot to the normal position, or draw the astragalus to a perpendicular line beneath the tibia. In order to further increase the mobility of the foot and thereby reduce the malposition, I drove my chisel in between the fibula and tibia until the lower fragment of the fibula was freely movable. I was then able, with the use of considerable force, to restore the foot to a normal position. The tip of the internal malleolus was then wired with two iron sutures to the tibia, (see No. 10). Two wire sutures were employed to approximate the fragments of the fibula (see No. 10). The foot was then dressed in the slightly inverted position, a plaster of Paris bandage being used as the outer retaining dressing. This extended above the knee. No unfavorable symptoms followed, and the foot was dressed in two weeks. The external wound was nearly healed. A week later, the patient was obliged to return to Texas. He directed the subsequent care and dressing of the ankle. A few days ago I received a letter from him, dated February 9, 1908, not quite nine months after operation, in which he says: " I began to put some weight on my foot about 2½ months after the operation. I am now able to walk without a cane and to attend to my business. The lateral motion and rotation are perfect. Flexion fair, with prospect of increase. It is perfectly straight, as you will see from the recent radiograph which accompanies this letter." (See Radiograph No. 10).

On February 17, 1908, I operated on the right ankle of Mrs. J., age 38, who had suffered from a Pott's fracture, which occurred one year ago, and resulted in such displacement outward of astragalus, with marked eversion of the foot, that walking or standing caused acute and continuous pain. Radiograph No. 12 shows the fragment of the tip of the internal malleolus carried outward with the astragalus, so it encroaches on

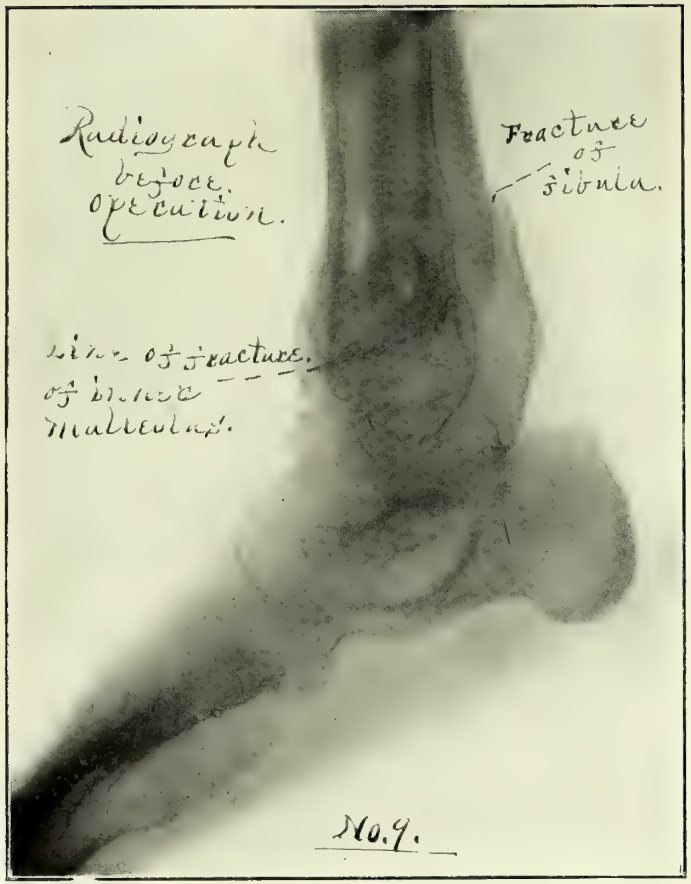


FIG. 9.—Before operation. Shows side view, with line of fracture of the inner malleolus and fracture of the fibula.
(Plimpton)

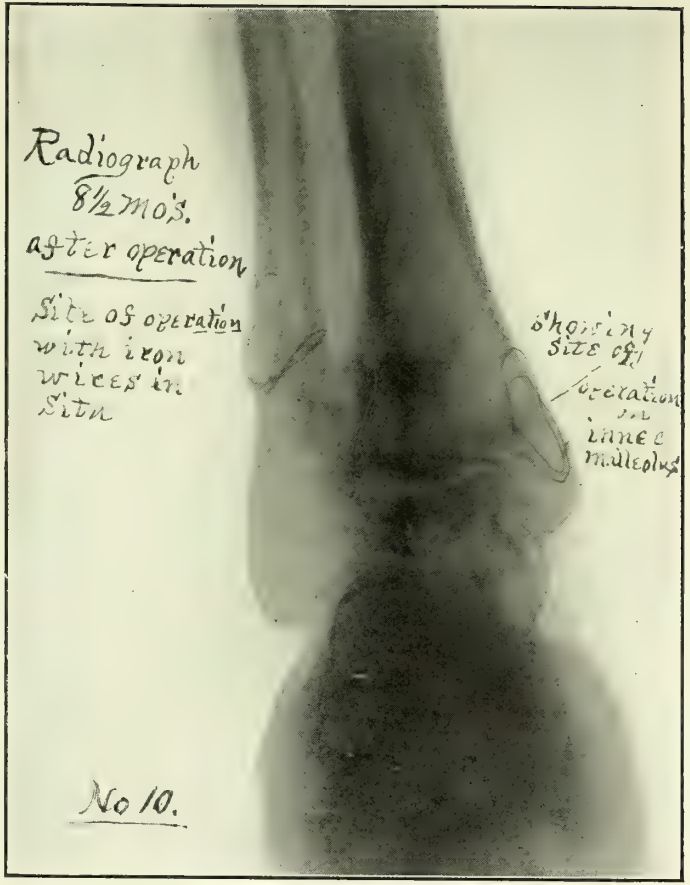
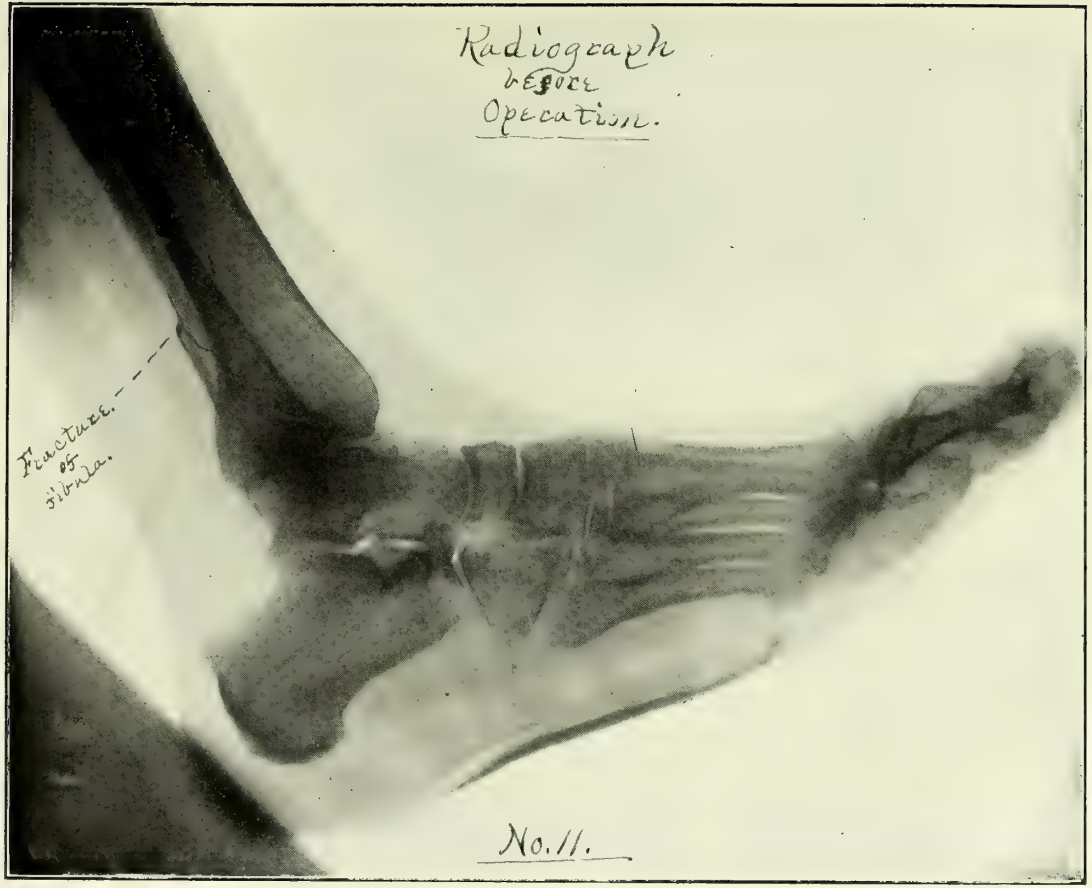


FIG. 10.—Taken 8½ months after operation. Shows sites of operation with good union, and iron wires still to be seen in situ.



(Plimpton) FIG. 11.—Side view. Shows fracture of the fibula before operation.

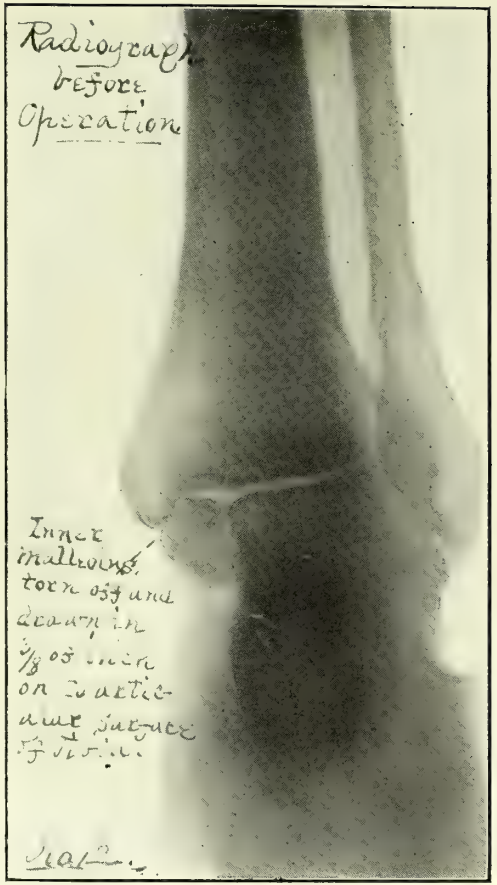


FIG. 12. Front view. Taken before operation. Shows the inner malleolus torn off and carried outward $\frac{3}{8}$ of an inch onto the articular surface of the tibia, with outward displacement and eversion of the astragalus.

the articular surface of the tibia about $\frac{3}{8}$ of an inch. I found an oblique fracture of the fibula about $2\frac{1}{2}$ inches above its tip, united by fibrous union. With chisel, I divided the fibula along the original site of the fracture, and divided the fibrous union of tip the of the internal malleolus. After forcing my chisel between the tibia and fibula to partially separate the two, I was able to restore the parts to their original positions. I then used four iron wire sutures, two on each side of the ankle, to retain the fragments in apposition as before, and dressed with the usual dressing, with the foot in the inverted position with the heel drawn down, and plaster dressing over all. Everything has gone on favorably, no temperature and little pain. I cannot yet report a further result, but expect it to be good. The foot is still in the dressing.

To summarize:—I have reported on 12 cases of deformity following Pott's fracture. In every one the operation for correction was on the joint itself. In every instance the result has been a strong and useful foot, satisfactory alike to patient and operator. The plan followed in operating has been to restore the foot to the axis of the leg, by bringing the astragalus squarely beneath the horizontal plane of the tibia By reproducing the mortise for its support, the normal relations of the leg and foot have been reestablished. While the operations have been different, they have aimed at the same principle in operating. In the early cases, a reproduction of the original conditions after fracture seems practicable. Late conditions must be treated more radically, if need be, by resecting bone and cutting of soft parts, until the foot can be again placed in line with the leg. Save the cartilage on one side of the joint in resections, if possible, to prevent ankylosis. Wire fragments when necessary, and, in short, remove or relieve obstructions as they appear in each particular case. The X-ray will assist, by pointing out what the operator will have to deal with, and aid him in formulating a previous plan of procedure. Suit the operation to the case at hand, with a clear idea of what you expect to accomplish, and you have the key to the situation.

ADVANCED MANAGEMENT OF CERTAIN ULCERATED CONDITIONS.

BY CARTER S. COLE, M.D.
Adjunct Professor of Surgery, New York Post-Graduate Medical School and Hospital.

There are always many conditions that offer problems difficult of solution, and there are others that have, for quite a time, baffled us and then ceased to be a source of much solicitude when we have found a uniformly successful method of dealing with them. It may be stated without fear of contradiction that, in a general way, malignant growths and ulcerations or other conditions resulting from malignant processes are far from being satisfactorily managed, with the possible exception of superficial epithelioma—where we seem frequently to be able not only to arrest but to cure the process by the use of terchloride of antimony (butter of antimony). While it was not my purpose to consider these lesions, except to emphasize the fact that they are not amenable to the treatment here offered for simple ulcerated conditions, I shall take occasion to mention them incidentally, and to give the technique of their care and cure. In using the term simple ulcerated conditions, I have given the limitations of the lesions to which the nutritive treatment seems especially applicable.

For at least twelve years, I have followed with unvarying success the principles and practice which I purpose to lay down; and this has not been done in the quiet and confidence of my own private work, but in full view of physicians from all over the world. In wrestling with the delay and deformities following more especially burns, the thought came to me that if the actual lesion could receive improved nutrition, we ought to get more rapid recovery and much less deformity of the surface involved. Extending this same idea to ulcers of the leg not due to syphilis—because in these an ointment of mercury seems to give them the food they crave—I was delighted to find that the same thing was true, and that ulcers that previously took months to get well would heal entirely in a few weeks. Not only so, but the marked improvement in nutrition would give a surface that seemed

to keep its covering with much less difficulty. Extending the same principle to loss and injury of bony surfaces, from tuberculous processes, like the shoulder and other joints, and using in such cases glycerinized extract of bone marrow, I have not been so fortunate, although improvement was more rapid than with the usual routine methods. It is unnecessary to state that diseased bony surfaces must be thoroughly curetted and the dead bone removed.

The idea is neither new nor original, yet it is surprising how many physicians seem never to have thought of this matter. Possibly, as a unit they are agreed that the use of cod-liver oil and other things on skin surfaces can produce constitutional effects; indeed, many think inunctions the best, if not the only way to use mercury. Others believe that the same method of administration of oleaginous substances in children is ideal. But if it be suggested that it is easier and more rational to get improved nutrition and more rapid healing by employing a food that the tissues can use *in loco*, they shake their heads.

Superficial burns that are very intractable and very uncertain, are those due to the X-ray and those due to a hot water bag. Of the deeper burns it may be said that those in the neighborhood of joints are apt to leave a cicatrix and to very seriously cripple the member. It is not necessary to remind the reader of the importance of always dressing such lesions with the limb extended, no matter what particular dressing be used. Generally, in any burn, an antiseptic dressing—preferably a one or two per cent. solution of carbolic acid—for forty-eight hours or more if the slough has not been removed by that time, should precede the nutritive treatment. Chemical burns are to be managed in the same general way, except that for the first two days an alkaline dressing in acid burns gives more relief, as a rule, than any other.

Scalds are often very difficult to cure. One of my earliest cases, in which the nutritive treatment proved of so much value, was a woman who had been treated at another hospital for several months. She came to my clinic with an extensive scalded slazy surface involving a part of the chest and left breast, the granulations apparently incapable of doing anything for themselves, and the wound as nearly a hopeless one as is generally seen in such cases. The nutritive treatment was in a few weeks followed by a most satisfactory cure, with a remarkably good surface and with very little cicatrization.

Ulcers of the leg (except tubercular, malignant and syphilitic), especially those in old people in whom the general condition is not good, must be converted into simple ulcers. This is done quickly and effectively by the use of a curette, not by poultices of any kind. It may be desirable to use an antiseptic dressing for a few days, though frequently this is unnecessary. It is often necessary to remove from the surface of the ulcer with a flat probe or the curette, a thin exudate that prevents any application, nutritive or otherwise, from reaching the granulations. Good support by a well applied bandage is always a necessity in every kind of ulcer of the leg.

Another class of rebellious ulcers are those of amputation stumps, and sometimes those on skin surfaces following some suppurative process. The same principle of treatment applies in these cases. I recall the hearsay case of a physician where a failure to cover after eighteen months, was in a few weeks entirely healed by the local nutritive treatment.

I have spoken of the fact that less cicatrization after deep burns follows this method of treatment. In support of this statement I recall an Italian whose forearm in the upper third, and the arm in the lower third were exposed to the deep fascia by a severe burn. To-day, in spite of a small unhealed area, flexion and extension are practically unimpaired. A short history of another interesting case must be added. A lady had suffered from a herpes affecting some branches of the fifth nerve. The lesions on her face became involved in an erysipelatous infection. Just what treatment had been used by several physicians who had been in attendance, I am unable to say; when I saw the case one side of the face presented a number of deep ulcerations that were most painful and disfiguring. An antiseptic dressing applied for a few days, followed by local nutrition, resulted in a most satisfactory cure. It is now almost impossible to tell which side of the face has been affected.

There is hardly a matter to which I have referred in which I could not cite a number of illustrative cases, but my effort is to be brief and at the same time not to be misunderstood. It is only fair to remind the reader that anything that will furnish nutrition to tissues will also make an excellent culture medium for bacteria, and a neglected nutritive dressing will provide more maggots in a short time than any other means

with which I am conversant. While I would not try to limit the list of possible nutriments, and while I have personally tried a number of things, the combination of water and alcohol, fat (ether extract), gelatine and albumen, peptone, creatine and meat extractives, salt, non-nitrogenous extractives and other mineral matters—commercially known as "Bovinine"—has been most satisfactory under such circumstances. In skin lesions I rarely use anything else. The glycerinized bone marrow, to which I have already referred, is still *sub judice* in the treatment of bone lesions. The technique is very simple. A piece of sterile gauze saturated with the nutrient solution is laid over the cleansed ulcer; over this a piece of rubber tissue or oil-silk, extending beyond the edges of the gauze, is used to retain the moisture and keep out contamination; over this a piece of dry gauze and a good bandage (to support the vessels and retain the dressing in the case of the leg, always begin at the foot) complete the dressing. This dressing should be changed daily or certainly at no longer interval than forty-eight hours. At this time the wound surface and its surroundings are washed clean with alcohol and the dressing repeated as before. The improvement is almost immediate. The gauze becomes white where it comes in contact with the ulcer, and is very little changed where it has come in contact with the skin. In an unusually short time —a few days—the progress is so definite that the patient will note the improvement.

I would like to give in detail a case of gangrene of the outer side of the right foot, extending to the arch and almost to the median line. The phalanges of the little toe were destroyed and a necrotic process involving a third of the metatarsal bone was present. Several excellent surgeons said that failure to amputate the leg promptly would result in the loss of the man's life. Two wanted to amputate above the knee and two below, the diagnosis having been made of gangrene due to endarteritis with thrombosis. The removal of necrotic bone, followed by a nutritive dressing over the area in which the tendons were exposed, resulted in a cure, which, for fifteen months at least, has given no concern.

In X-ray burns I have used local nutrition from the first flush, a short time after exposure, and in the most intractable ulcerations, which had resisted treatment for a long time, and

always with the most satisfactory results. In fact, if I were deprived of the nutritive method in the treatment of X-ray burns, I could not promise a cure by any other method with which I am familiar.

I spoke of epithelioma, the superficial variety in skin surfaces, and I mentioned a drug long known for its value in such lesions but frequently forgotten, viz: terchloride of antimony. The surface is dried, not scraped, cut or curetted, and the solution (10%), which was formerly officinal, applied. Over this a wet antiseptic dressing (2% carbolic) or a dry sterile dressing is applied. Fresh applications are made as often as a scab forms on the skin which can be lifted off without tearing the base of the ulcer, generally at intervals of three or four days. The cervix cases in early stages of ulceration (epithelial degeneration) have responded most satisfactorily to repeated applications of this drug; at least it can be tried before more radical and destructive measures are employed.

To summarize: In simple ulcerated conditions which are slow to heal, or refuse to heal, we have in local nutrition an effective and expedient adjunct to their cure. In superficial epithelioma we have the terchloride of antimony, a valuable drug to control and to relieve and to heal the ulceration.

PRIMARY EPITHELIOMA OF THE VAGINA IN A PATIENT UNDER TWENTY YEARS OF AGE.

BY GEORGE GRAY WARD, JR., M.D.

Professor of Diseases of Women, New York Post-Graduate Medical School and Hospital.

The following case was referred to me by Dr. F. W. Best, of Port Jervis, N. Y., and was admitted to my service at the Post-Graduate Hospital on February 13, 1908.

The history was as follows:

Edith P. was 20 years of age on January 21, 1908. Born near Middletown, N. Y. Has been married for four years, but has never been pregnant. Her menstruation is regular every 28 days, duration three to four days, and without pain. She has never had any illness or injury prior to this trouble, and her weight last summer was 170 pounds. Her present illness dates from October, 1907, when she first noticed some vaginal discharge, which was tinged with blood. Dyspareunia was also becoming marked. The discharge later became offensive and defecation was painful, and, as she was losing weight and becoming debilitated, she consulted her family physician. As it was thought advisable for her to see a specialist, she came under my care.

On examination the patient presented the appearance of a strongly built country girl, who was unusually pallid. She stated that she had lost 20 pounds since October last. A careful inquiry could elicit no evidence of syphilis. The family history was negative. As to carcinoma, the possibility of the patient's disease being tubercular in nature, was in mind; and a careful inquiry made as to tuberculosis in the family was likewise negative. Her husband, who accompanied her to the hospital, was of frail constitution and of a tubercular type. He was in fair health, but had had pneumonia last winter. Examination of the patient showed the heart and lungs to be normal. The abdominal organs gave no signs of disease. Her temperature was 99°, pulse 88.

The external genitals were those of a nulipara and perfectly normal. On exposing the vagina with a retractor there was revealed an area of deep ulceration of the entire posterior vaginal wall from just in front of the cervix to the site of the

hymen, and which extended half way up each lateral vaginal wall. The area of ulceration was rectangular with sharply defined straight borders, as though a knife had been used to outline them. The edges were hard and infiltrated, and the rectal wall was completely exposed, and covered with a grayish slough with a necrotic odor. The inguinal glands were not involved, and on deep palpation the pelvic glands could not be made out. Rectal examination showed that there was as yet no perforation, but the outer coat of the bowel was becoming infiltrated.

The uterus was of normal size and in position; the broad ligaments were apparently not involved in any way, and the appendages were normal to touch. The cervix presented a normal appearance with no evidences of laceration. There was a slight reddening at the external os.

The examination was conducted under anesthesia and was made with care, but no evidence of disease could be found other than on the posterior and lateral vaginal walls.

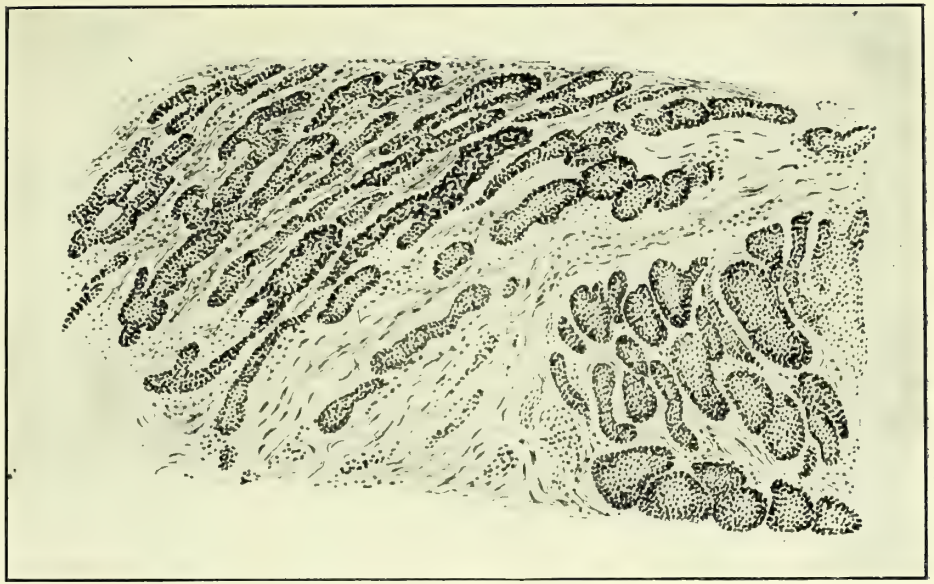
The cervix was dilated and curetting obtained from the uterus for microscopical examination. Sections of the infiltrated margins and of the base of the ulceration were removed and sent to Professor Brooks for a report.

A tentative diagnosis of primary cancer of the vagina was made, and the disease was considered as having advanced too far to allow of an operation being done with any advantage.

The surface and margin of the growth were cauterized with the actual cautery to clean up the slough, and the vagina was packed with iodoform gauze. While waiting for the pathologist's report, and in view of the husband's physical condition, the tuberculin eye test was applied through the kindness of Dr. Dunham, but it was negative.

The following report on the specimens was received from Professor Brooks:

"Microscopic examination of the tissue removed from the margin of the ulcer situated in the posterior wall of the vagina of Edith P., age 20 years, received February, 13th 1908, showed narrow bands of loosely reticulated fibrous connective tissue containing a moderate number of spindle-shaped nuclei. This tissue was arranged in the form of elongated, ovoid alveoli of quite uniform size, which were more or less densely filled with



(Ward) Infiltrating epithelioma of the vagina. ×105. Leitz 3, oc. IV.

small cells of the squamous type, with large nuclei and a small amount of cystoplasm. The cells were more densely arranged at the periphery of the nests, next to the fibrous stroma; and at the center there was occasionally seen a space resembling somewhat a lumen. The general features of the structure corresponded to epithelioma. The histologic arrangement is well illustrated in the accompanying figure, which is a faithful reproduction of the characteristic portions of the growth.

The uterine curettings showed no histologic deviation from the normal."

As the diagnosis of primary epithelioma of the vagina was confirmed and thus a hopeless prognosis inevitable, on account of the inoperability of the case, I felt anxious to try the effect of radium on the growth, especially as this was a cancer of the pavement epithelial type.

Through the kindness of Dr. Morton, four tubes of radium, each containing 10 mg., were employed in the treatments, which were carried out under the direction of Dr. Sturges. The tubes were placed in line so that the radium was distributed throughout the length of the posterior vaginal wall, and the exposure lasted for one hour. Exposures were made on February 26, 28, and March 2. Careful examinations made up to the present writing, March 22, revealed no change in the growth except that of steady progression.

The patient's condition is rapidly becoming worse; cachexia is now marked, emaciation is progressing, and she is in constant pain which requires increasing doses of opiates. Perforation has occurred, and the present treatment is that of cleansing and deodorant douches, with iodoform gauze packing.

Frequency.—Primary cancer of the vagina is a disease of great rarity. H. McNaughton Jones has recently written to a number of continental observers and has received the following replies as to its frequency:

Fritsch, during 13 years in the Bonn Frauenklinik, had not seen a single case. In Breslau he had seen four cases. Kroenig, during two and a half years at Freiburg, had not seen a case. At Jena and Leipzig he could remember having operated upon only four cases, and with one exception the disease quickly recurred. Jacobs at Brussels had met only four cases in all his experience. Bumm, at Berlin, has seen only one case in 433

cases of carcinoma of the female genitalia. Schöttlander, at Heidelberg, has found four cases in 150 cases of uterine cancer. This is above the usual average. Franz, at Jena, has seen two cases in 125 cases of carcinoma of the portio since October, 1904. Zweifel, at Leipsic, has seen four cases out of 1400 cases of carcinoma, up to 1906. Friedl states that, up to 1896, the journals contain records of 130 cases, and Cumston, of Boston, says there are only 150 cases up to 1900.

W. Roger Williams, in a study of this disease, states that of 9226 tumors in women, 54 were in the vagina. Of these 40 were carcinoma, two sarcoma, three myoma, and nine cystoma. Therefore, he believes that cancer is by far the most common form of primary vaginal neoplasm, in spite of its rarity. He has seen five cases. His data, from London hospitals, show only 0.43 per cent of all cancers in women to be of vaginal origin. Reiche, in Hamburg, tabulated 7498 deaths from cancer, and of these only 29 were vaginal, or 0.38 per cent. Hecht in 4507 cases found 50 vaginal, or 1.1 per cent. Gurlt in Vienna gives 1.6 per cent. Walshe in 3076 cases found 14 were vaginal. Martin gives the proportion at 1 per cent. Bergel found 14 cases in 8287 deaths by cancer. Schwarz at Berlin found 0.24 per cent. and Pozzi 0.42 per cent. Bernard states that there were two cases out of 475 women in Pozzi's service. Schmideleschner says recently, that in many thousands of cases of cancer observed in his clinic during the past 23 years, he has seen but seven cases of primary vaginal epithelioma. Eppinger, in the pathological Institute of Prague, has encountered 79 cases of cancer of the uterus and three of primary origin in the vagina. Williams has averaged many of these observations, and he gives the percentage of vaginal origin as less than 1 per cent. This rarity is certainly astonishing when we remember that the uterus, a connecting organ, is most prone to the disease of all the organs in women. Zizold states that this is due to the anatomical construction of the vagina, it being deficient in glands.

Age.—Epithelioma of the vagina is essentially a disease of adult and post-meridian life, but in numerous instances it occurs at an earlier age than cancer in general. Williams has combined 17 cases which he has studied to Bernard's 59, making a total of 76 cases, and has found the age distribution was as follows:

20 to 30 years	.....	12 cases.
30 to 40 "	.....	14 "
40 to 50 "	.....	12 "
50 to 60 "	.....	22 "
60 to 70 "	.....	14 "
70 to 80 "	.....	2 "

In Williams' 17 cases, the average age of onset was 40 years. (In uterine cancer it is 44 years and in mammary 48 years.) The earliest age of onset that he has found was 17 years.

The classical cases, frequently reported in the literature, of Guersant, 3½ years, Johannovsky 9 years, and Smith 14 months, must almost surely have been sarcomatous. Sarcoma of the vagina is much more frequent in children than in adults. Starfinger studied 26 cases of sarcoma of the vagina and found 24 were under five years of age. The majority of the cases, according to the above table, arise between 50 and 60 years, or after the menopause.

Trapenard, in a more recent study (1907), found somewhat different results. He has tabulated 40 cases and found the ages as follows:

20 to 30 years	.....	2 cases.
30 to 40 "	.....	15 "
40 to 50 "	.....	10 "
50 to 60 "	.....	7 "
60 to 70 "	.....	4 "
70 to 80 "	.....	2 "

He therefore found the predominance between 30 and 50 years. Küstner has collected 24 cases, and the majority were between 30 and 40 years of age; two were between 15 and 20, and two between 20 and 30. Gebhard has reported a case in a girl 14 years old.

Pregnancy.—Pregnancy seems to have no bearing upon the etiology. Nuliparæ are as prone to the disease as multiparæ. In one-fourth of Bernard's cases there had never been a pregnancy. Küstner found it in six virgins.

Heredity.—Heredity seems to have little bearing upon the etiology. Küstner found hereditary cancer once in 22 cases. Pozzi reports one case where both parents died of cancer.

Baldwin reports a case of the father and grandfather having cancer, and Fenger saw a case where the grandfather and aunt died of the disease. Trapenard, in his 40 cases, found it clear in but one.

Irritation.—There are but few cases reported where any mechanical irritation or trauma could be considered as a contributing cause. Hegar reports a case following ulceration due to a pessary. Remarkably few cases are reported where there has been vaginal prolapse and invagination.

Site.—The disease may start from any part of the vaginal walls, but in by far the greater number the origin is on the posterior wall. In Bernard's and Williams' cases, 50 arose from the posterior wall, 15 from the anterior wall, in nine the lateral walls were first affected, and in two the disease was annular. Olshausen reported 18 cases, 13 of which involved the posterior wall.

Pathology.—The vagina resembles the skin in its histological structure far more than it does a mucous membrane. It is lined by stratified epithelial cells, arranged as in the integument. There is an absence of hairs and glands, and, according to Williams, a decided absence of pigment, which accounts for the extreme rarity of the melanotic types of cancer in this location. While the greater surface of the vagina is covered with pavement or squamous epithelium, cylindrical cells are present in the ducts of Gartner and in Skene's glands. Epithelioma of the vagina in its structure resembles epithelioma of the integument, and it nearly always originates in the squamous epithelium. It is usually classified into two varieties, the lobular and the tubular, according to the arrangement of the cells. The lobular variety is the most frequent. It has been demonstrated by Reclus that the plaques of leucoplakia met with in chronic vaginitis may become transformed into epithelioma.

Polosson and Violet describe a form of cylindrical epithelioma which originates from the remains of the ducts of Gärtner, and state that the theory of congenital anomalies explains this type. The cases of grafted neoplasms observed by Hartmann explain the origin of certain cases of squamous epithelioma.

Macroscopically, the tumor shows two forms. The most frequent is that of a papillary growth. The second form is that of an infiltration. The papillary form is usual to the pos-

terior vaginal wall, while the nodular or infiltrating form is more frequently observed on the anterior wall, in the neighborhood of the urethra. It sometimes occurs as a general annular infiltration of the whole tube of the vagina.

The first manifestation of a primary vaginal carcinoma is usually a single nodular or papillary mass. Cases have been reported, however, of primary multiplicity. Olshausen has seen a case of probable "contact infection," the disease being propagated from the posterior to the anterior wall. Ulceration rapidly follows the appearance of the growth, with a tendency to spread towards the vulva rather than the uterus. The pelvic and inguinal glands are usually soon involved. Metastases are not common.

Symptoms.—The symptomatology is the same as for carcinoma elsewhere, viz.: bloody discharge, foul necrotic odor, pain, and cachexia.

The average duration of life in Williams' cases was 16.5 months; the longest was 26.25 months and the shortest eight months.

Prognosis.—The prognosis is very grave and recurrence is the rule after operation; exceptions are noted, however, as instanced by the case recently reported by Legueu, where he operated for a papillary epithelioma involving the posterior vaginal wall ten years ago, and his examination at the present time shows no evidence of a return of the disease. All observers are in accord that pregnancy greatly hastens the disease.

Conclusions.—Primary epithelioma of the vagina has been subjected to careful study by a number of observers, notably Bernard, Bonnefons, Polosson and Violet, Peyrache, Trapenard in France; Küstner, Brückner, Hirsch, Olshausen and Zizold in Germany, and Roger Williams in England, besides many others who have contributed to the subject. There are probably many cases which are not reported.

The case I report is of unusual interest, first on account of the extreme youth of the patient, she being but 19 years and 8 months of age when the disease was first noticed, and second on account of the type of the epithelioma, it being of the infiltrating type, on the posterior wall, which is not so common as on the anterior; and third, on account of its being treated by radium.

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TWENTY-FIVE YEARS IN MEDICINE.

BY REYNOLD WEBB WILCOX, M.D., LL.D.
Professor of Medicine in the New York Post-Graduate Medical School and Hospital.

As one reviews a quarter of a century and its work in the practice of a special branch of the art of healing, only landmarks along the road traveled recall themselves. These may not be the really important ones, unless one takes himself from his daily routine of important matters and petty details and adjusts his perspective. In all recollections the personal element must obtrude itself. The most lasting impressions are, doubtless, those made by our early teachers, for through them we lay the foundations for future careers. These facts, doubtless, represented the best in medical knowledge of that period. Fitz, in pathology; Shattuck, since a professor, in diagnosis, then mostly physical and practically limited to auscultation and percussion, Minot, in theory and practice, with Ellis teaching the clinical aspects of medicine, and Edes in therapeutics, essentially pharmacal, were all men of broad culture, substantial information and scientific enthusiasm. Nothing was presented but in accord with the highest professional ideals, and represented the best of American teaching. Looking back upon it from the standpoint of present century ideals, one may say that the foundation was substantial in pathology, in diagnosis and in theory; but the guiding hand of the master in application of these principles leading to a therapeutic conclusion, which should be of the greatest value to the patient, was not to be found. The training was the best of its time, but to-day more is demanded.

In Vienna, the work of Bamberger and Duchek, and to a less extent that of Mynert, was followed and considerable personal experience was acquired and practical information obtained; but treatment was either routine or skeptical, and the result to the patient came chiefly in verifying pathological diagnoses and adding to the collection of morbid statistics. Pathology seemed to occupy so much of medical thought and diagnosis less, with treatment almost a negligible quantity, that this branch was again seriously taken up with Arnold, at Heidelberg,

and the foundations relaid. Here, also, Freidreich, building well with his colleagues' basis, inspired real enthusiasm in diagnosis which was often exhausted before treatment was reached. In Paris, however, a different picture presented itself; treatment began to receive attention. The teaching of Seé, Dujardin-Beaumetz, Charcot and Hayem; the abundant opportunity for laboratory and clinical studies, having for their end the treatment of patient and disease and, so far as drug therapeutics was advisable, the aid of elegant pharmacy, resulted in relegating to the background pathology and diagnosis, not because they were unimportant but because they were essential only as a foundation for improving the condition of the patient and combating disease. The despair of the dead-house which had resulted in German therapeutic nihilism, here was supplanted by a French optimism which was for the good of the patient. In Edinburgh, it was expected that treatment would occupy the centre of the stage, as, indeed, it did; but the work of Stewart, Wyllie, and Fraser did not reach the ideal of modernity after the experience of the preceding two years. It was by no means time misspent, for much was learned of bedside management of disease.

In the year 1884, the pupil became the teacher; facts which he had laboriously acquired in the dead-house, the laboratory, the dispensary, and the hospital, were now to be utilized for the benefit of mankind. Those who had trodden similar paths were to be aided in practical work, to the end that they might practice their profession in a way more acceptable to their patients and in harmony with a peaceful conscience. For nearly three years the Post-Graduate Medical School had been gathering under one roof successful teachers of medical graduates who had been teaching in clinics and hospitals in which they held appointments. The medical schools viewed the growing number of matriculates with dismay. That the physician could need more than they could impart, was incredible. That their teaching was not fully abreast with medical knowledge, was impossible; it was an experiment destined to fail, because its success was predicated upon their failure to fully equip their students for professional lifework. The faculty, many of whom knew from personal experience the shortcomings of medical instruction, with foresight remarkably clear as it was accurate

provided the means for remedying the defects. The increasing number of matriculates demonstrated the accuracy of the convictions of the faculty and efficiency of their plan to meet the demand for better medical practice. One direct result has been that medical schools are now teaching diagnosis in satisfactory manner, although their efforts in the line of treatment leave much room for improvement.

The past twenty-five years have been of remarkable fruitfulness in medicine and especially in therapeutics. The old humoral pathology had declined and the cellular was accepted only to yield to and be modified by that of the infectious. The pathogenic bacteria were being recognized and their importance in diagnosis appreciated. Later came studies of their toxins which, in logical sequence, the antitoxin followed, their action modified by the alexins and the phagocytic theories made more tenable by work upon the opsonins. The great impetus to direct medication and its possibilities to be reached in the near future, are even yet hardly realized. The old doctrines of elimination were supplemented by inhibitory action, even when brought about by chemical means. The further advance in this direction was the demonstration of the intermediate host and the prevention of many infectious diseases.

Opotherapy, resulting from the solution of the mystery of the ductless glands, has borne fruitful results and diminished the number of hitherto supposed to be incurable diseases. Even now much remains to be learned. The studies of the chemistry of digestion, begun in France, elaborated in Germany and carried to ultimate analysis in this country, have given an impetus to investigation of the broader problems of metabolism which has already yielded practical results. The diathetic diseases are now approached with considerable mathematical precision. The blood, in diagnosis especially, yields much information from which pertinent conclusions may be drawn. The excretions, indicating quite accurately end-results, have furnished much confirmatory evidence of previously accepted theories. During this period medicine has been reclassified and a new nomenclature has arisen.

In pure therapeutics, the development of the science of physical chemistry and, in particular, the ionic theory has reduced medication to more nearly an exact science. The resources

of the pharmocological laboratories have been utilized, not only in the development of new synthetics, but in demonstrating the varieties of drug-action and offering suggestions for broadened uses. Physical therapy, cultivated with renewed ardor, has enlarged the resources of the clinician and established itself as of real value. The Roentgen ray, of importance in medicine second to its use in surgical diagnosis, offers yet unappreciated possibilities, and this is even more true of other forms of light and even of radium. Electricity has yielded brilliant results, even if in a limited field. Climate and exercise no longer rest upon an empirical basis.

These years of productive activity have yielded tangible results, not only in diagnosis, but especially in treatment. Diphtheria has now a mortality of one-third that of former days, enteric fever one-ninth, pneumonia one-sixth; while in many diseases their duration has been reduced from one-half to one-seventh of their previous ascribed periods. Considerable numbers of incurable diseases now yield to modern treatment or are absolutely cured. The health-asset of the country has been multiplied many fold by the prevention of diseases, especially of those of an infectious character. All of this enormous advance in medical science and much more that suggests itself to the thoughtful reader of scientific literature, has come about during the life of one medical generation. The commercial value of the medical man to-day to the community is beyond computation.

It has been justly said that the architecturally pleasing buildings of the New York Post-Graduate Medical School and Hospital, are a monument to Daniel Bennett St. John Roosa. His clear insight into the future, his abiding confidence in the profession, his undaunted courage in overcoming obstacles, his implicit trust in those teachers whom he gathered about him, and his unerring judgment, made the school not only a thing to be desired, but, as we see medical science now, a necessity. Not only must we admire his remarkable work in his chosen department of medicine, of which he was a distinguished exponent, but as well his influence upon every teacher in the faculty, inciting all to earnest endeavor for science and its benefits to matriculates and patients. Himself a writer of terse and vigorous scientific English, he counted among his associates twenty-

five authors of forty-two volumes and three translations. The inspiration of Roosa as a noble man and an accomplished scientist, has influenced the faculty to put forth their best endeavors and, through them, the twelve thousand matriculates of the school, for the benefit of hundreds of thousands of patients in their struggle for health and even for life itself. The appreciation in which he is, and will be, held by all who came in contact with him and by those who, in turn, in a wider circle have been aided by his life, constitutes a monument more important and enduring than a material one raised by the hand of man.

URETHRAL CALCULUS.

BY JOHN J. MCGRATH, M.D.
Professor of Operative Surgery, New York Post-Graduate Medical School and Hospital.

James B., age 24 years, entered the Harlem Hospital upon the suggestion of Dr. Frederick E. Bauer, December 14, 1907.

The patient stated that four weeks before admission he began to experience difficulty in urination, delay in starting the flow and diminution in the volume of the stream. The difficulty gradually increased until, for the past few days, the urine has been discharged in drops and only with considerable straining and some pain. Finally, for the past 16 hours he has been unable to pass any urine whatever. Previous to the beginning of his present trouble, dating back four weeks, he claims that he has had no symptoms of any kind referable to the bladder or urethra. He had never passed any blood.

Upon admission the patient was found to be suffering from retention of urine. The bladder was very much distended. An attempt to introduce a catheter into the bladder was not successful, and a metal instrument met with an obstacle which prevented its passage and which communicated a distinct, gritty sensation and click. A hard lump, the size of a pigeon's egg, could be felt in the perineum just back of the scrotal fold. This mass was not adherent to the skin and was not especially tender.

Under ether anesthesia an incision was made in the middle line of the perineum down through the mass, and a stone was removed which is represented in the accompanying illustrations (Fig. 1). The stone was found lodged in the bulbous portion of the urethra. The wall of the urethra was much thickened and swollen and fitted snugly around the stone. After the stone was removed, a sound was passed from the incision in the perineum, through the deep urethra, into the bladder. No further stones were detected in the bladder. The calibre of the deep urethra accommodated a No. 30 French sound, and was certainly not sufficiently roomy to permit the passage of such a large stone as the one which had been removed from

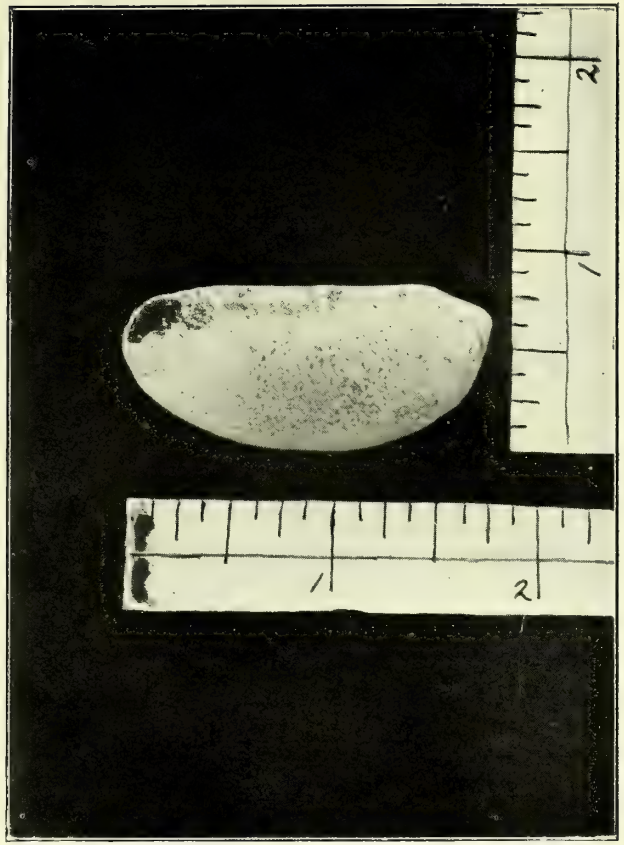

FIG 1.—Showing dimensions of the calculus.
(McGrath)

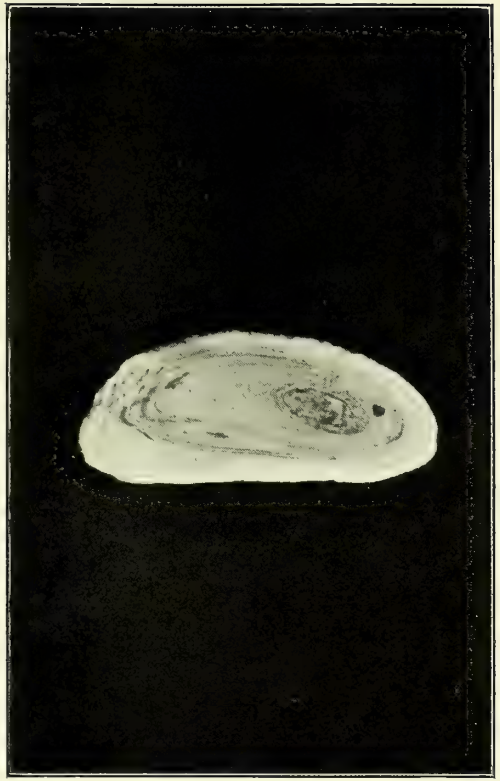

FIG. 2.—Section of the stone showing its layer formation and uric acid nucleus.

the bulbous portion. The patient was discharged from the hospital cured, December 30, 1907.

Vesical calculi are not infrequently driven from the bladder into the deep urethra and, if not too large, may traverse the entire length of the urethra and thus escape. Larger stones usually become impacted, completely blocking the urethral canal, and cause acute retention which continues until relieved by a surgical operation. In the case, the history of which is given above, the stone had evidently been lodged in the urethra for some time and, apparently, without causing any discomfort, to judge from the patient's story. It probably escaped from the bladder as a small uric acid stone, as represented by the nucleus seen in the illustration of the cut section of the stone (Fig. 2). It passed through the prostatic and membranous portions of the urethra and became arrested in the comparatively roomy bulbous portion. In this location it remained without causing any urgent symptoms, permitting the urine to flow past it, between it and the urethral wall, ball-valve fashion, gradually increasing in size and assuming the form of a cast of the bulbous portion of the urethra by the deposit of phosphates, until it reached its present size and, finally, a slight increase in the swelling of the mucous membrane of the urethral canal was sufficient to completely block the canal and shut off the escape of urine.

An interesting feature is the tolerance of the urethra to the lodgement of a calculus for the considerable period necessary to have resulted in the large stone exhibited. It could not have escaped from the bladder in its present size and, according to the history, it did not begin to cause inconvenience until four weeks before operation, when it had probably acquired sufficient size to seriously obstruct the urethral canal. How long before this time the stone in its original form had entered the urethral canal, is uncertain. There is nothing in the history to determine this time.

Examination of the stone, section and photographs were made in the laboratory of the Post-Graduate Hospital under the direction of Professor H. T. Brooks, who reports that the nucleus of the calculus is composed of uric acid, and the outer layers of calcium carbonate, calcium phosphate and ammonio-magnesium phosphate.

FAULTY METABOLISM AS A CAUSE OF CHRONIC INTERSTITIAL NEPHRITIS.

BY FREDERIC E. SONDERN, M.D.

Professor of Clinical Pathology, New York Post-Graduate Medical School and Hospital.

At the time when the etiology of chronic interstitial nephritis was quite obscure, the disease was seldom recognized until a well established pathological lesion of the kidney had resulted, and recovery was and is still considered quite impossible when the condition has advanced to this degree.

The general belief now exists that the actual pathological lesion of the kidney is invariably preceded by a period of months or years during which clinical investigation will reveal increased arterial tension and abnormal heart action. The urine at this time shows the evidences of a functional albuminuria and not those of a contracting kidney. The etiological factors of these premonitory signs are now considered to be general arteriosclerosis on the one hand, or some form of faulty metabolism on the other. The development of this usual type of nephritis as a part of a general arteriosclerosis in old persons lends the impression of general senile change.

The other general causative element, namely, faulty metabolism, has been the subject of much investigation, as the result of which it is to be hoped that chronic interstitial nephritis may be prevented in many instances.

In order that this may be brought about, it is necessary that greater attention be given to faults in body chemistry, and that existing ones be remedied not only during a short period of subjective ailment. The necessity for permanent change in habits and mode of life, in order to remedy these faults, is the reason why they are so seldom eliminated. Perusal of the causes of this type of nephritis, quoted by most authors, indicates that faulty body chemistry must be the common one, and it also seems reasonable to assume that general arteriosclerosis, at least that occurring prior to advanced age, may also rest on this basis. Diatetic influences, functional disorders of the liver, lithemia, gout, worry and strain of business, overeating and lack of exercise, are but other names for faulty metabolism. As the causative condition quoted would indicate, the fault is

usually found to be improper nitrogen metabolism and the seat of this fault is probably in the liver. To go a step further would be to claim that a hepatotoxemia exists.

A clinical laboratory experience of about twenty years in private practice presents quite a number of cases from which specimens of urine were minutely examined, at longer or shorter intervals, over periods of 19 years, 15 years, 10 years and five years, and these examinations justify the belief that intestinal toxemia is also a potent factor in the etiology of the type of nephritis under consideration.

Unfortunately, the available time did not permit search for records of all cases of chronic interstitial nephritis in which the condition of the urine has been followed for at least five years and including a period prior to the development of the evidences of an actual pathological lesion of the kidney. In one case 35 urine examinations have been made during the past 19 years. The early records show no abnormal features, the analyses having been made as a matter of precaution before anesthesia and before application for life insurance. Just ten years ago, with insignificant symptoms referable to the gastro-intestinal tract, the urine contained a large amount of indican and showed a disturbed sulphate ratio but no fault in nitrogen excretion. During the next two years three specimens were examined with the same result. For the following five years the urine presented evidences of a functional albuminuria, together with the indications of faulty metabolism noted. Three years ago the classical urinary picture of the disease—large daily amount, pale color, low gravity, deficient solids, traces of albumin and few casts—developed with the usual clinical symptoms. Since then the patient has given his condition the strict attention it should have had at an earlier date. Proper climate and mode of life have caused all evidences of toxemia to disappear, but the pathological process continues. It seems reasonable to assume that intestinal toxemia was the direct cause in this case.

In one case observed over a period of 15 years the urine was normal for two years, presented evidences of faulty nitrogen metabolism without albuminuria for ten years, with functional albuminuria for two years and classical urine and symptoms of the disease for one year. Death resulted at the end of this time with positive autopsy record.

Four cases, observed for ten years, presented evidences of

faulty metabolism at the time of the first examination—one, intestinal toxemia and three, faulty nitrogen metabolism. In these cases the period prior to actual evidences of nephritis was also invariably long.

In 22 cases observed for five years, functional albuminuria and evidences of faulty metabolism were present at the time of the first examination; intestinal toxemia in seven; hepatotoxemia and faulty nitrogen partition in 12; both conditions combined in three. All these cases have developed the classical urinary picture of chronic interstitial nephritis. There were no evidences of general arteriosclerosis in any, as far as could be learned.

The small series quoted would seem to demonstrate faulty nitrogen metabolism in the majority, and intestinal toxemia in a considerable proportion. In reading the histories of the comparatively rare cases of chronic interstitial nephritis in children, the impression is invariably gained that faulty metabolism existed. Either there is a record of long standing gastro-intestinal derangement, or one which justifies suspecting the presence of a socalled lithemic tendency.

While it must be admitted that faulty metabolism seems a potent factor in the etiology of chronic interstitial nephritis, the question arises, why a large number of persons suffering from severe grades of faulty metabolism, for very long periods, never develop the disease or show evidences of functional albuminuria. It seems probable that unknown hepatic or intestinal toxic poisons in the circulation are the substances which cause spastic contraction of arterioles, vascular hypertension, cardiac and renal change, and that these faults are *not* due to the presence in the blood of abnormal amounts of normal constituents, the result of faulty body chemistry. Ordinarily, the degree of hepatotoxemia or intestinal toxemia is judged approximately by the degree of the resulting faulty body chemistry, but this is, by no means, an invariable rule. Cases are not infrequent in which faults in body chemistry can be demonstrated and no evidences of toxemia found, and, on the other hand, decided evidences of toxemia occur when the faults in body chemistry are disappointingly slight. The subject will remain obscure to the scientist and unsatisfactory to the clinician until the time when these toxins are isolated, their origin determined and methods for their detection and estimation described.

THE SURGICAL TREATMENT OF "UNRESOLVED PNEUMONIA."

BY SAMUEL LLOYD, M.D.

Professor of Surgery, Post-Graduate Medical School; Visiting Surgeon Post-Graduate Hospital and Babies' Wards.

Unresolved pneumonias are frequently spoken of by medical men without any very satisfactory explanation of the reason why the pneumonia persists instead of pursuing its natural course and undergoing resolution at the usual time. Jackson, in the *Boston Medical and Surgical Journal* some two or three years ago, in studying this subject called attention to the fact that an unresolved pneumonia very seldom exists, and Musser says he has never seen a case to which he could apply this term. On the other hand, a good many writers have referred to the persistence of pneumonia and have described the symptoms without giving any explanation of the pathological conditions that seem to fully explain the symptoms.

Increasing experience with any operative technique necessarily gives the operator better judgment in dealing with the conditions, enabling him to observe closely and accurately conditions which would be overlooked by the inexperienced. It has fallen to my lot within the past twenty years of active service in the wards of the Post-Graduate Hospital, to operate upon about 500 cases involving the removal of ribs and the opening of the pleural cavity, and yet it is only within the past two or three years that I have reached any possible explanation of the socalled unresolved pneumonias.

One of the first cases to come under observation after I had begun to suspect what might be an explanation of the condition, was a man 47 years of age who, some years before, had been admitted into St. Francis Hospital with a pneumonia which had failed to resolve, and who was finally supposed to have tuberculosis involving the lower lobe of the right lung, although no tubercle bacilli were found in his sputum. As he had proven a reliable ward attendant, he was retained in the hospital for several years, having occasional attacks of pain, fever, and depression which

invariably cleared up on a free escape of mucopurulent secretion, which he expectorated in considerable abundance. Finally, it was determined that he had an encapsulated empyema. This was operated upon and drained, leaving a sinus in the lower part of the chest—about the 8th rib—from which there was a more or less constant discharge of muco-pus. Although I had seen him around the wards for several years and had known that from time to time he had these periods when he was confined to bed, I supposed it was an ordinary tubercular case and that nothing could be done for him except the ordinary routine treatment which he carried out himself.

About two years ago, however, the sister in charge of the surgical ward asked me to see him with a view to determining whether he could be improved. I then found that whenever he had these attacks of illness, the external sinus had closed and his temperature, pain, and cough returned and persisted until the sinus was reopened or he coughed up a large quantity of pus.

I operated upon him, removing about three inches of the 7th and 8th ribs, gaining free access to a cavity in the pleura about the size of an ordinary orange. In examining the pulmonary surface, I was able to make out a depressed sinus dipping forward into the lung and opening into a cavity about the size of an English walnut. Irrigation of this cavity allowed the fluid to run through the bronchus into the mouth, demonstrating the fact that he undoubtedly had a pyopneumothorax; the pus in the pleural cavity was localized by adhesions between the pleuræ, and the bronchus terminated in a small abscess cavity in the lung itself. I immediately broke down the adhesions between the pulmonary and the parietal pleuræ—making sure that the lung remained in full expansion by the method which I shall describe later and which has been published in the *Annals of Surgery*, March, 1907—and then cut away the external wall of the pulmonary abscess, curetting the remainder of the cavity with a sharp spoon to make sure that no limiting membrane was left to interfere with the proper cicatrization of the pulmonary tissue. This patient made a complete recovery and left the hospital.

A second case was one I operated upon in the Post-Graduate Hospital about a year ago and reported in a paper published in the *Medical Review of Reviews*, February, 1907. This child

had been under treatment for an unresolved pneumonia and had finally developed an empyema occupying the upper half of the chest. At the operation, instead of getting the usual clear pus, the discharge was brown and very offensive. The lung followed down after the removal of the fluid, so that the lower portion, which was already fairly well expanded, fully occupied its position in the chest; the apical portion also expanded as the limiting adhesions were released. The upper portion of the lower lobe, however—the empyema was on the left side—did not expand so well, the result being that the lung had the appearance of a dumbbell. On passing my finger over this contracted area I found that it was solid, and expressed the opinion that it was an area of unresolved pneumonia. The cavity was irrigated with normal saline solution, and, while the fluid was pouring in and out of the cavity, a section of this contracted lung—about three inches long by an inch and a half in width—dropped off into the pleural cavity. I immediately seized this piece and on examination found that it was a gangrenous portion of the lung. The space left in the lung was interesting, because it demonstrated that the same thing happens in pulmonary tissue as in other tissues where gangrene is localized. Free oozing of blood could be seen over the whole of the open surface. No hemorrhage of any moment occurred, and a few washings of hot saline solution were sufficient to control all the oozing from the raw surface. I could not see that the exfoliation of this piece of lung had the slightest effect upon the child's condition. During the first two or three days after the operation there was a decided amelioration in the symptoms, and at one time I even hoped that, in spite of the grave septic condition, the child might recover; but she died of sepsis on the sixth day.

Shortly after operating on this case, another one was referred to me from the medical department of the Babies' Wards, with an obscure lung condition which was supposed to be an unresolved pneumonia. Aspiration of the chest did not reveal any fluid, but there was marked flatness about the level of the 4th or 5th rib behind. An X-ray revealed the presence of an abscess, and I excised the 6th rib, taking out about three inches along the posterior axillary line. A little serous fluid was found in the pleural cavity and a few adhesions to the anterior chest

wall just above the line of incision. Inspection immediately revealed the fact that the interlobar space was obliterated by adhesions. These were broken down, and an abscess about the size of a small hen's egg was evacuated.

Two weeks ago a man was admitted into my wards, transferred from the medical side of the Post-Graduate Hospital, with a history of empyema following pneumonia. In this case, at the operation, we obtained the same foul-smelling, brownish fluid that was found in the case of gangrene of the lung already described. The lung was firmly adherent to the anterior chest wall, and at first did not expand; but after carefully breaking up the adhesions, the apex and upper portion of the lower lobe expanded satisfactorily; the lower portion of the lower lobe was still flat and not well aerated. Careful examination of this area showed a gangrenous patch over the posterior portion, with a navel-like depression at about its centre, which communicated with the bronchus. We were able to demonstrate here a large slough not yet separated from the lung. The man's condition has improved since operation, and under daily saline irrigations the offensive odor is disappearing and shreds of tissue are being washed out. He is making a satisfactory convalescence.

On May 4, 1907, in consultation with Dr. L. E. La Fetra, I saw a boy, about seven or eight years of age, who had had a pneumonia followed by empyema, which had been operated upon by Dr. La Fetra on the third of April. The pneumonia had begun on March 17. Immediately on opening the chest cavity, the temperature fell to normal and then gradually rose until there was a range of between 102° and 103°. During this time the pus had decreased in quantity, but there was a large amount of mucus being thrown off. When I saw him on the third of May, I concluded that there was an opening from the bronchus into the walled-off space in the pleura and that the lung was incompletely expanded. I enlarged the opening and broke up all the limiting adhesions, allowing the lung to fully expand, and was then able to demonstrate to Dr. La Fetra and the others present at the operation, the opening through the lung into the bronchus. In the course of a few days the discharge became almost purely bronchial mucus, exactly corresponding to that coughed up during the course

of a bronchitis. This was examined by Dr. F. E. Sondern, who corroborated the bronchial type of the discharge. Several applications of nitrate of silver—10 to 20 grains to the ounce—were made through the chest wall into the bronchus, and the child was discharged well on the sixth of July. Dr. La Fetra tells me that he has since been through a severe attack of bronchitis without any signs of a return of his pulmonary fistula.

Within the past year I have seen two or three other cases of a similar character in my service in the Babies' Wards of the Post-Graduate Hospital, but those already quoted suffice for the purpose of this article.

Unresolved pneumonias, in my opinion, are rarely, if ever, medical cases, but are evidences of destruction of pulmonary tissue that require surgical treatment. It seems to me that an explanation of this condition may be somewhat as follows: First, the pneumonia, follows its usual course; then, either a fresh infection or the pneumococcus taking on a pyogenic character, an abscess develops in or about the pneumonic area. This perforates into the bronchus, with the escape of a large amount of muco-pus through the mouth in coughing and a gradual clearing up of the symptoms, or through the pleura into the pleural cavity, producing a general or a localized empyema. In cases where the pneumonic area lies close to the inter-lobar space, the abscess may be encapsulated in this region and may give the impression of a localized pneumonic area.

Recently I operated, at the Post-Graduate Hospital, upon a case of empyema in which pneumonia was still present. The lung was firmly bound down against the posterior chest wall and did not respond to the respiratory efforts. On loosening the adhesions and expanding the lung, an encapsulated abscess was found in the inter-lobar space and in the upper portion of the lower lobe (the pneumonia was on the left side.) We could distinctly see the pneumonic area running along practically the whole superior surface of the lower lobe posteriorly, and in a state of gray hepatization. In this case, in order to free the lung from the diaphragm, it was necessary to cut the adhesions with a pair of scissors. If the infection is very intense, gangrene of the lung results. All cases of unresolved pneumonia should be examined first with the X-ray, and if the skiagraph shows a shadow at the point

where the pulmonary symptoms have been made out with the usual auscultatory signs, then a good-sized exploring needle should be inserted and the presence of pus determined, if possible.

Early operation with liberation of the localized pus foci in the lung, will undoubtedly avert more extensive damage by preventing perforation into, and infection of, the pleural cavity, or breaking down of a large abscess cavity in the lung itself. One of the cases that I have operated upon for an abscess of the lung, and which I have not included in this report, has since developed undoubted signs of tuberculosis and is still under observation in the Babies' Wards. Had his disease remained confined to the lung, I should have operated upon him again, but the appearance of a kyphosis about the seventh dorsal vertebra demonstrated the fact that he has other tubercular foci. I have not, therefore, considered it wise to proceed further surgically in his case.

In order to handle these cases properly, it is essential to have a well-defined technique; to overcome the fear attendant upon operations on the chest, and to recognize the fact that almost all of the statements made in the text-books in regard to the collapse of the lung when the chest wall is opened, are based upon improper physiologic observations.

If the lung is allowed to collapse when a pulmonary abscess is present, it is impossible to locate the abscess unless it is of large size and has no communication with a bronchus. When the lung collapses it presses the contents of the abscess into the bronchi so that it escapes through the mouth, leaving no indication of the point where it existed. This was well illustrated in a case that I operated upon in the Post-Graduate Hospital for Dr. Leonard Weber—a young girl who had inhaled a tack and had developed an abscess in the upper part of the lung. I had no difficulty in getting down through a good free opening over the area where the X-ray revealed the abscess, but when the chest cavity was opened the lung was completely collapsed and the anesthetist called our attention to the free escape of pus from the mouth. I carefully palpated the lung but could not discover the abscess. In this case my technique was at fault; I should not have allowed the lung to collapse. In operating upon an abscess of the lung now-

a-days, I take the precaution to keep up full expansion throughout the course of the operation. It is then often possible to see a bulging on the surface of the lung indicating the abscess area, or to recognize even a small perforation in case it has already broken into the pleural space and produced empyema.

All adhesions in the pleural cavity that interfere with full expansion of the lung should be thoroughly broken up. In making this statement I feel that I am on safe ground. The number of operators who avoid breaking down the adhesions around an empyema for fear of infection of the general pleural space is fortunately growing smaller every year, and it is now advocated and practiced by so many good observers, as is evidenced in the current medical literature, that I do not consider myself called upon to defend this portion of the technique of my operation.

A great deal of experimental work has been carried on in the last four or five years to develop some method of operating upon the lung without allowing collapse of that organ to take place. The cabinet devised by Sauerbruch, in Miculicz' clinic, at Breslau, and the work of Brauer, at Heidelberg, are well known; and in the *Annals of Surgery*, of February, 1908, there is an interesting paper on experimental surgery of the lungs, giving a preliminary report of thirty animal operations under positive pressure. All of this work is absolutely unnecessary if the proper technique is followed. Not only can the lung be made to expand in any human being, in spite of an opening in the chest wall, but it can be kept in full expansion throughout even a prolonged operation, or can be made to collapse at the will of the operator.

We have been able to demonstrate this so many times during the past fifteen years in our clinics at the Post-Graduate Hospital and in my service at St. Francis, and so many men have seen the actual demonstration of the method, that I feel perfectly safe in making this assertion in the emphatic way that I do. Unfortunately, all of this other experimental work is undertaken because the idea has become so fixed in the minds of the medical profession that if an opening is made in the chest wall larger than the diameter of the main bronchus, collapse of the lung must result. This is absolutely true if no pre-

cautions are taken to prevent such collapse, because the atmospheric pressure is naturally greater than the intra-thoracic pressure. It is also absolutely true if both pleural cavities are opened at the same time.

In order to carry out the plan that I have outlined it is evidently essential that the lung and pulmonary pleura retain their elasticity and capacity for expansion. It is also necessary to demonstrate that expansion can take place without the application of any external positive pressure, as suggested by Robinson and others, or without the complicated cabinets for the application of negative pressure suggested by Sauerbruch. The pleura must also retain its irritability.

The Estlander and Schede operations were devised at a time when it was supposed that any interference with a lung that was bound down could only result disastrously to the patient. They believed that in empyema, the pleural space having been converted into an abscess cavity, the only way to close that cavity was by getting an agglutination of its surfaces. All the rules of surgical procedure up to this time were opposed to interference with the lung itself, and as every one was convinced that a lung once collapsed must always remain in that condition, the only possible method of closing the cavity was to remove the bony structures over a sufficiently large area to allow the two pleural surfaces to come in contact. The Fowler and Delorme operation was a tremendous step forward. These operators, however, were still bound by the idea that if a pleura had been thickened and contracted by the formation of an abscess over its surface, it had lost all its elasticity and would not allow of expansion of the lung itself. They therefore advocated decortication of the lung, recognizing the fact that the pulmonary tissue itself was capable of re-expansion if the limiting pleura was removed. In this way they made a distinct advance over the Estlander-Schede methods, because they filled the cavity by expanding the collapsed lung and avoided the mutilation which was the necessary result of the older methods of operating. Ransohoff's method, published about a year ago, is only a modification of the Fowler-Delorme operation, and was developed because of the same idea that the pulmonary pleura was the cause of the non-expansion of the lung. All these operations are developed along lines of false

observation. The pulmonary pleura is just as capable of re-expansion as the lung tissue itself, and there is no necessity in any case of empyema of performing decortication in order to obtain proper expansion.

It is evident, therefore, that my plan of operation depends upon certain facts which are contrary to the usually accepted ideas—some of them so radically different from accepted teaching as to require demonstration to carry conviction. In the first place, a collapsed lung can be made to expand until it fills the pleural cavity without the aid of any artificial means, no matter how large the opening in the chest wall may be.

In the second place, the lung can be made to expand without any interference with the pulmonary pleura.

In the third place, the success of the operation depends upon utilizing the irritability of the pleura itself, which has never failed to respond in any of the cases that I have operated upon.

The operation should be carried out in the following way: Having demonstrated the presence of pus in the pleural cavity, a point between the anterior and posterior axillary lines should be selected for the incision. In determining upon the site for the opening in the chest, we must take into consideration the necessity of breaking up the adhesions throughout the whole of the pleural cavity, and must select a point that will enable us to reach the apex and at the same time free the lung from the diaphragm. The location of my incision, therefore, is no longer at the bottom of the pleural cavity, but is as nearly as possible midway, and on the side rather than in front or back. In small children, three inches of the 6th rib will usually answer; in adults, it may be necessary to take a three-inch piece out of the 5th, 6th, and 7th, to enable the operator to reach all parts of the pleural cavity easily. As soon as the ribs have been excised—and this is done subperiosteally—the pleura is opened. At first it is best simply to make a small puncture, allowing the pus to escape gradually, until it is evident that the withdrawal of pressure from the pleura will not disturb the heart's action. The anesthetic must be ether. The patient is put completely under and kept in complete anesthesia until the ribs are excised. As soon as this is done the anesthetic should be stopped and no more administered unless during the course of the operation it should become necessary to collapse the lung again. As soon

as possible the pleural opening is enlarged to its full extent and the finger seeks out the location of the collapsed lung. Sometimes it is found in the apex of the cavity, contracted down like a rubber ball; at other times it spreads out along the back, firmly adherent to the posterior wall and to the diaphragm and pericardium, so that it is a thin, ribbon-like structure. At first sight it seems impossible to obtain any improvement in such an organ; but, feeling the way carefully, the finger separates the adhesions, keeping as close to the parietal pleura as possible during the manipulations. If the empyema is recent, the adhesions break just as readily as about the appendix in a recent case of appendicitis; if it is old, it may be very difficult to separate them and at times almost impossible to recognize the line of cleavage between the diaphragm and the lung. During the course of this first exploratory examination, the coagulated lymph will be loosened from its attachments and can be drawn out of the cavity; if it is not readily disposed of, hot normal saline should be poured from a pitcher into the pleura until all of these coagula have been washed away, in order that they may not interfere with the sense of touch or with the view of the expanding lung as it begins to come forward. If still large masses adhere, they should be scraped away with a sharp curette. If the adhesions are so firm that they cannot be broken with the finger, they should be cut with a pair of curved scissors as close to the parietal surface as possible. It is true that in this separation of the adhesions we frequently tear into the lung tissue itself and sometimes even into a bronchus. One need have no fear of complications in case this accident occurs, as the hemorrhage is usually very slight and stops quickly when the cavity is irrigated; or, if one is not certain about how much bleeding is coming from the tear, the anesthetist may reapply his ether and bring the patient again under complete anesthesia, when the lung will again collapse and the operator can examine the size of the laceration of the lung tissue. In case there is any bleeding formidable enough to require control, the spot may be touched with the Paquelin cautery, or a purse-string stitch may be passed around and tied. When this is done, the anesthetic should be again withdrawn and the breaking of the adhesions continued. If the adhesions are very extensive, it

will be found from time to time that there is considerable blood clot in the cavity, and this had better be washed out with hot saline poured from a pitcher as before.

During separation of the adhesions, the patient is gradually recovering from the effect of the anesthetic, and it will soon be discovered that as the finger sweeps the pulmonary pleura the patient will develop a hacking pleuritic cough. With each cough the collapsed lung expands until it not only fully occupies the cavity but often will be pushed through the opening and portions of it can be drawn outside. This is readily explained. The irritation of the pleura causes a sudden expulsion of air from the healthy lung through a partially closed glottis. The obstruction is, therefore, at the upper end of the breathing apparatus; more air is being driven through the trachea than can escape through the superior opening, and the surplus must flow over into the open bronchus of the collapsed lung. In other words, we compel the patient to use the healthy lung as a pump to blow up the collapsed one.

Following the publication of my description of this operation in the *Annals of Surgery* last year, I received the following letter from Mr. Bilton Pollard, Surgeon to the University College Hospital, in London.

24 Harley Street, West,
London, England, April 6, 1907.

Dear Sir:

I have just received the March number of the *Annals of Surgery*, and have read with much interest your paper on the treatment of empyema. I feel sure that you will be interested also in the enclosed papers from which you will see that I have been guided by the same principles as yourself in the treatment of empyema. It is now twenty-one years since I began working (at the Northeastern Children's Hospital, London) on the lines laid down in the paper. I have, since I wrote in 1901, gone further in the matter of breaking down the adhesions than is urged in the paper, but I must freely admit that I have never pushed my views in the matter of adhesions to their full logical extent, as you have done. There is no doubt that the right time to mobilize the lung is at the original operation for empyema, and I trust that the congratulations of a colleague who has worked on similar lines will be acceptable to you.

Faithfully yours,

Bilton Pollard.

Mr. Pollard's article appeared in the *Clinical Journal*, London, February 27, 1901, page 293. I cannot help quoting

from this article, because it is such a remarkable coincidence that we should have expressed ourselves in almost the same words. As I said, in my article in the *Annals of Surgery*, my first explanation of the actual mechanical reason for the expansion of the lung was cleared up for me by a conversation with Dr. A. H. Smith, on the evening of November 11, 1891, and since that time I have always explained the matter as I did in the article referred to. Mr. Pollard says:

"Every one must have noticed in making a digital examination of the pleural cavity after opening an empyema that at first the lung is collapsed and possibly only just within reach of the finger. Perhaps, after making this observation, the patient happens to come out of the anesthesia sufficiently to cough. Now, what happens? If the finger is in the pleural cavity at the time, it will feel the lung expand, and in many cases come into contact with the chest wall. The explanation of this is obvious. The ribs fall together and the arch of the diaphragm rises during the violent expiratory effort. By this means the size of the pleural cavity is diminished, but that is not all. The collapsed lung is itself inflated. During the cough the glottis is closed and is not at once forced to open by the air which the expiratory effort drives from the healthy lung, for there is a free vent for it from the opposite bronchus into the collapsed lung. The glottis being opened, air is drawn in from outside, and subsequent coughs drive more and more of it into the collapsed lung and expand it. Supposing now that the anesthesia is deepened. The lung shrinks somewhat but does not collapse to anything like its previous state. It is, of course, well known that it is well-nigh impossible to squeeze the air out of a lung post mortem, and in the condition of things we are contemplating the only force which can act upon the lung is its own elasticity. But there is, I think, during life, another condition which assists to maintain the expansion of the lung. I refer to the blood pressure. Surely this, acting on the innumerable capillaries of the air cells, would produce a condition which I would liken to a number of springs which would together exert a considerable force on the side of expansion. But even if my argument be freely admitted so far, there still remains something to be explained. After the cessation of coughing, although the lung does remain fully expanded, it nevertheless shrinks sufficiently to leave an interval between it and the parietal pleura, and rapid healing by adhesion between the two parietal surfaces must require actual contact. A simple experiment which I have often made will lead us a step further. After an empyema has been freely opened, air passes in and out of the cavity readily during inspiration and expiration. If whilst the opening is uncovered the chest be auscultated, little or nothing can be heard of the breath sounds, but if a dressing be applied and the chest be again auscultated, after a few minutes' intervals the breath sounds will be more audible and if the patient coughs a few times they may often be heard quite distinctly.

Now what is the explanation of this? As long as the air can enter the pleural cavity freely the enlargement of the thorax during inspiration exerts no influence on the lung. When the opening is closed by a dressing, the entry of air is more difficult and then the expansion of the chest acts upon the lung and air is drawn in through the bronchus, and audible sounds are produced. After a time, too, the meshes of the deep dressing become filled with purulent discharge, and then the dressing acts as a sort of valve. During inspiration it offers a considerable resistance to the entry of air into the pleural cavity. During expiration, and particularly during the expiratory effort of coughing, air is expelled from the chest and escapes beneath the edge of the dressing. Owing to the coughing the lung is now blown out, sufficiently to come into contact with the parietal pleura over a considerable area, and a sort of sucker action is established—just as a piece of wet leather tightly pressed against a stone cannot be separated without considerable force, so the lung remains in contact with the chest wall."

He says there are three clear indications for treatment: to break down as far as possible the adhesions between the lung and the chest wall; to rub off the lymph from the surface of the lung; and to use some means for distending the lung and so to break down or stretch loose the adhesions.

I have quoted thus fully from Mr. Pollard's paper because, in the first place, he has expressed some points more clearly than I have ever succeeded in doing, and because I had not thought of the suggestion he makes in regard to the blood pressure; and, finally, that I might express my thanks to him for his courtesy in sending me his corroborative views in this line of work.

It will be readily seen that, with the gradual expansion of the lung during the liberation of the adhesions, the surgeon cannot fail to recognize whether the aeration spreads throughout the whole lung or whether there are areas which remain flat and offer more resistance to the examining finger. When this latter condition is present ocular investigation should be added to the sense of touch. A pair of retractors should be inserted on each side of the incision, the wound spread open to its full extent, and the free fluid remaining in the cavity from previous flushings taken up with gauze sponges until the pulmonary pleural surface can be clearly seen. It will often be recognized that some area will have a grayish appearance, and palpation at this point sometimes reveals a sense of fluctuation. Not infrequently when an empyema is present, a rupture will be

seen over this surface through which pus can be pressed. If this condition is recognized, the opening should be enlarged, stretching it with a pair of forceps until the cavity in the lung is exposed. This cavity can then be readily curetted with a dull curette and, if necessary, wiped out with a piece of gauze on a pair of forceps. If no opening is present, the pulmonary pleura is seized with a pair of catch forceps. It is necessary to use catch forceps in this procedure, because the lung acts freely with each respiration and the successive expansions and retractions pull the pleura away from an ordinary pair of thumb forceps. The pleura is then incised with a pair of scissors and the opening dilated by stretching, until it is free enough to allow the escape of pent-up secretion. I have never seen any hemorrhage greater than a mild oozing, and the opening of the bronchus interferes only slightly with the expansion of the lung itself. The oozing is soon controlled by washing with hot saline, and then, if a piece of gauze is held firmly over the opening in the bronchus, the lung can be made to expand to its full extent.

The success of this operation depends first, upon the evacuation of all the pus; second, upon the removal of all the lymph coagula, even curetting these away if necessary, so that the pleural surfaces may fall in contact when the full expansion is obtained; third, breaking down all limiting adhesions; fourth, irritation of the pleura to induce coughing; fifth, washing out of the cavity to get rid of all blood clots and debris.

The washing-out of the pleural cavity is, of course, open to criticism. The bad results that formerly followed irrigations of the pleura after operation for empyema have created an almost insuperable prejudice against this procedure. I can readily see that filling the chest cavity with fluid while the lung is still bound down, pouring it in through a small opening, and not allowing for sufficient egress, will increase the pressure to such an extent that serious consequences result; but I never allow ordinary irrigation of the pulmonary cavity. The wound must be held well open and the fluid poured in from a pitcher; only so much fluid then enters the chest as can fill the spaces about the expanding lung, and the active motions of the lung during respiration swash this fluid around and force it out again through the opening. There is, therefore, never any increased hydro-

static pressure exerted, and the hot solution seems to have rather a stimulating effect upon the patient.

We soon found that the insertion of a drainage-tube in the pleural cavity after the lung was thoroughly expanded, kept up a constant irritation of the pleura, and caused a persistent hacking pleural cough. In some instances this was so serious that it exhausted the patient and necessitated removal of the tube. On this account the spool drainage-tube, which we now use, was made for me from suggestions made by Dr. H. D. Furniss, then House Surgeon of the Post-Graduate Hospital, and Dr. F. N. Wilson, Instructor in Surgery in my department. These tubes are made in different lengths to fit the varying thickness of the chest wall. In this connection, it is interesting to find that Mr. Pollard has also been using a tube of exactly the same shape and size. He writes me that this tube was figured and described in the twelfth edition of *Heath's Minor Surgery*, " revised by me in 1901, and about the same time it was described in the *Lancet* or *British Medical Journal*, and made for general sale by Mayer and Mellzer, of London, in nine different sizes. I had used it several years before."

As Dr. Furniss' suggestion was made in 1901, it is evident that Mr. Pollard's use of the tube antedated its use in our clinic.

In the early part of the paper I said that ether was the anesthetic that should be used in these cases. The reason for this is that the recovery from the ether is more gradual than from chloroform, and consequently there is more time for intrathoracic work before the patient becomes fully conscious. It is essential that the anesthesia shall cease at once after excision of the ribs and before the pleura is opened. This should be done, because occasionally we find that the fluid is evacuated very rapidly in spite of all attempts to control it, the intrapleural pressure being so great that it bursts through the pleura as soon as the soft parts are cut through and the ribs removed. In these cases, if the adhesions are recent, the lung breaks away from them, fills the pleural cavity, and at the same time a large amount of the anesthetic is inhaled. A more important reason for stopping the anesthetic is to allow the patient to recover consciousness during the remainder of the manipulation.

12 West 50th Street.

CARCINOMA OF THE HEART.

BY T. HOMER COFFIN, M.D.

Instructor in Pathology, New York Post-Graduate Medical School and Hospital.

Although medical literature concerning diseases of the heart is extensive, there are few references to malignant tumors of this organ. The reason for this is two-fold: firstly, malignant tumors involving the heart are of rare occurrence, and, secondly, these tumors, when present, rarely produce symptoms which lead to a diagnosis of the condition.

Tumors of the heart may be primary or secondary, benign or malignant. The malignant tumors are sarcoma and carcinoma, which are almost always secondary.

Primary Heart Tumors.—Primary cardiac tumors are very rare. Nearly all forms of mesoblastic tumors have been found in the heart. The most common are fibromata, lipomata, myxomata, rhabdomyomata and cysts. Myoxmata usually occur in the auricles as nodular, glossy tumors. Rhabdomyoma usually forms multiple tumors growing upon or beneath the endocardium, the cells of which sometimes resemble ganglion cells. Congenital primary tumors have been described. Thorel,[1] in 3000 autopsies, found no tumors of the heart which he could call primary. Berthensen,[2] in a series of 28 primary tumors of the heart which he collected from the literature, found nine sarcomata, seven myxomata, six fibromata, two lipomata, three carcinomata *, and one cyst. In regard to the situation of the tumors he found that seven involved the right auricle, three the right ventricle, seven the left auricle, and five the left ventricle.

Secondary Heart Tumors.—Secondary tumors are found in the heart more frequently than primary tumors. They are metastatic, and grow from emboli of tumor cells which lodge among the trabeculæ of the heart, in the valve segments or in the coronary arteries. Tumor cells have been demonstrated in the heart blood by von Recklinghausen, Rieder, Weber, Lücke,

*These "carcinomata" are referred to below. No microscopical examinations were made; it is quite certain that they were not carcinomata.

Paulich, Birsch-Hirschfeld and others.[3] Tumors may involve the heart by continuity from tumors of the esophagus, mediastinum, stomach or lung. The secondary deposits may involve the endocardium, the myocardium or the pericardium.

Frequency of Cardiac Carcinoma.—That carcinoma of the heart is rare is shown by Bodenheimer[4] in his comprehensive dissertation. He states that Forster, Eppinger and Eiselt computed that 7.5% of cases of carcinomata with general disseminated metastases involve the heart. Pic and Bret[5] state that the ratio of secondary carcinoma found at autopsy is about 3 in 1000.

The following table, arranged by the writer from figures given by various authors, illustrates the frequency of carcinoma involving the heart:

Authority.	Number of Autopsies.	Number of Secondary Carcinomata.	Remarks.
Chambers[6].....	2161	7	
Willigk[7].....	4547	9	477 carcinomata in various parts of body.
Kohler[8].....	9118	6	
Pic and Bret[5]....	1708	25	
Thorel[1].....	3000	15	No primary cardiac tumors.
Johns Hopkins Hospital[10].....	2942	10	One primary tumor.

The occurrence of primary carcinoma in the heart is disputed. Bodenheimer claims that this may be the case, but Libert denies their existence.

Symptoms.—The physical signs of cardiac neoplasm vary. They depend upon the size and position of the tumor. Large endocardial tumors may cause cardiac insufficiency, by preventing closure of the valves, or they may obstruct the flow of blood by their size. If extensive in the myocardium, they may produce the symptoms of broken compensation. Bodenheimer states that the symptoms are usually produced mechanically. There may be precordial pain, a sense of oppression, anxiety, dyspnea, dizziness, edema of the extremities, effusion into the serous cavities, syncope, etc. Ribbert states that the heart may be involved so extensively with metastases that only a small portion of the muscle is left. Lister wrote of a meta-

static tumor in the right cavity of the heart which clinically produced a systolic murmur at the apex. Lenoble described a case with multiple metastases in the heart which clinically produced in the vessels of the neck a murmur like the purring of a cat. One of Deguy's[11] cases had heart crises like angina pectoris. Napp[12] describes a case, a man 42 years old, with carcinoma of the stomach, in whom a nodule, measuring 5.5 x 3.5 cm. was found in the apex of the heart. This nodule had so extensively replaced the muscle that normal myocardium was present only at the apex. The lumen of the right ventricle was so narrowed that there was only a small slit for the passage of blood. Although there was this marked obstruction, the heart tones and action were normal to the time of death.

Report of Case.

Epithelioma of the esophagus with secondary involvement of the myocardium, lungs, liver and kidney.

S. B. Hospital number 1002, aged 38, was admitted to the Surgical Wards of the New York Post-Graduate Hospital complaining of difficulty in swallowing. He had been unable to take solid food for about two months, and had lost strength and weight rapidly in the three months prior to admission. He complained of cough and dyspnea which had persisted for about three weeks. No blood had ever been expectorated. The symptoms, while the patient was in the hospital, were chiefly dyspnea and dysphagia, though the pulse-rate was accelerated (112 to 124) and the respirations were from 36 to 38 to the minute. Examination of the sputum showed numerous pneumococci, staphylococci, few streptococci and no tubercle bacilli. The sputum was abundant, purulent and had a foul odor. The patient died two days after admission. The autopsy report is briefly as follows:

The body is quite emaciated; rigor mortis present to a marked degree; the epitrochlear and cervical lymph glands are palpably enlarged.

Esophagus.—Palpably thickened at the level of the bifurcation of the trachea, and, on freeing the surrounding tissues, it is seen to be about two or three times its normal size, the enlargement beginning at the bifurcation of the trachea and extending almost to the cardia. On opening the esophagus the lumen is markedly

stenosed at a point 30 cm. from the teeth. The growth begins 4 cm. above the stenosis. The tumor has involved the esophageal wall so that at its thickest portion the esophagus measures 3.25 cm. in diameter at a point 22 cm. from the teeth. The tumor is soft and friable, yellowish white in appearance, and there has been some slight ulceration on the mucous surface of the esophagus overlying the tumor mass.

Heart.—The pericardial sac is normal. The heart is normal in size, but soft and flabby in consistency. The myocardium is pale. On the epicardial surface of the left ventricle, lying in the course of the coronary artery, about 1.5 cm. from the apex, is a small, yellowish white nodule 5 mm. in diameter. On section this nodule is moderately firm and has apparently invaded the myocardium to some extent. Microscopic examination shows the nodule to be made up of an embolus of epithelial cells, which has completely occluded the lumen of the coronary artery. This metastatic growth has invaded the myocardium and the muscle fibres near it are degenerated. (See Fig. 1).

Lungs.—The left lung is adherent to the chest walls throughout its entire extent. The apex is involved in an extensive abscess, the cavity of which occupies almost the entire upper lobe. The contents of this abscess are chocolate-colored and have a very offensive odor. Microscopically, the abscess-wall shows areas of carcinomatous growth. The lower lobe is markedly congested, but not consolidated. The bronchial lymph glands are enlarged and firm. Upon section they are yellowish white and apparently involved in the metastatic process. Microscopically, they show carcinoma. The right lung is practically normal.

Liver.—The liver weighs 1800 grams, is firm in consistency, and pale in color. In the right lobe there are numerous nodules, resembling metastases, varying from 3 mm. to 1 cm. in diameter. Microscopically, they show carcinomatous structure.

Kidneys.—The kidneys are normal in appearance, but in the cortex of the left kidney there is a small nodule, measuring about 2 mm. in diameter, which is apparently metastatic. Microscopically it shows carcinomatous structure.

In this case the cardiac metastasis was shown in microscopic sections to have occurred by the formation of an

embolus of epithelial cells, which had lodged in the coronary artery near the apex of the heart. The extensive carcinomatous involvement in the lung was probably the origin of this embolus. Hektoen, Siegel-Delval, and others have described cases similar to this one. Emboli from the lung may reach the heart: (1) through the pulmonary veins to the left auricle and ventricle, and pass into the coronary arteries, or, (2) through the bronchial veins, vena azygos and superior intercostal veins to the subclavian, to the vena cava, right auricle and ventricle. The position of the openings of the coronary arteries, just above the cusps of the aortic valve, offer opportunities for emboli in the heart blood to enter these vessels. The photomicrograph (Fig. 2) shows the mass of carcinoma cells in the coronary artery.

Carcinoma of the Esophagus.—Coplin[13] states that carcinoma of the esophagus is rare compared with carcinoma in other parts of the body. Tanchon,[14] in 9116 cases, found only 13 of the esophagus. Other writers state that 5% of carcinomata involve the esophagus.

Carcinoma of the esophagus is usually of the squamous epithelial type. Butlin,[15] in 54 cases, found three scirrhous, one medullary, one colloid; the remainder were squamous. The growth manifests a tendency to become annular, and the infiltration of the esophageal walls with the tumor cells causes proliferation of the connective tissue, resulting in thickening and loss of elasticity. As a result of this process, symptoms of obstruction, accompanied by dilatation above the stricture, develops. The mucous membrane covering the growth usually ulcerates.

The most usual site of carcinoma of the esophagus is the lower third of the tube, at the junction with the left bronchus. Other portions which are naturally narrow are sometimes the seat of malignant growths. The tube is narrowest at its commencement, opposite the sixth cervical vertebra and lower border of the cricoid cartilage, 15 cm. from the teeth. It is somewhat contracted behind the left bronchus, 26 cm. from the teeth, and at its passage through the diaphragm, 37 cm. from the teeth. In 901 cases collected by Kraus,[16] 45 involved more than one part of the organ, 397 the lower third, 302 the middle, and 158 the upper third.

FIG. 1.—Showing carcinomatous embolus in left coronary artery.

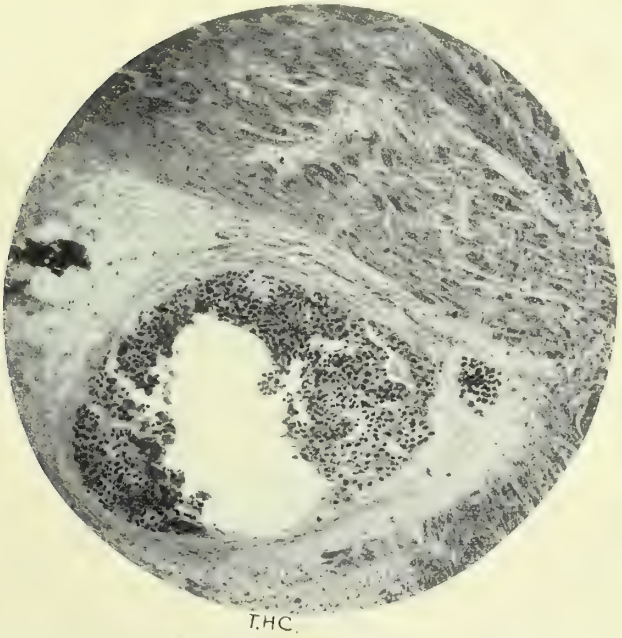

FIG. 2.—Embolus of epithelial cells in the coronary artery. From this embolus the myocardium became involved in secondary epithelioma. (Coffin)

By extension, the neighboring organs may become involved, causing various complications. The following are some of the interesting cases in the literature illustrating this point: Biggs[17] reported a case of carcinoma of the esophagus in a patient who had been unable to swallow food for six months. A bougie showed a stricture 12 inches from the line of the teeth. Later, at operation, the stricture was found 3.5 inches from the cardiac orifice. The patient died from hemorrhage following rupture of the aorta, which was involved by the growth. At autopsy the growth was found to begin at the bifurcation of the trachea, extending 8 cm. below this point. There was an opening into the aorta, measuring 2 mm. in diameter. Davidsohn[18] reports a similar case in which erosion of the aorta caused fatal hemorrhage. The growth in the esophagus was at the point of crossing of the left bronchus. Adenot and Cadet[19] described a case of esophageal carcinoma in which erosion of the growth caused an esophago-tracheal fistula, with resulting gaseous distention of the stomach.

CARDIAC CARCINOMATA DESCRIBED IN THE LITERATURE.

Boether[20] collected 71 malignant tumors of the heart from the literature, which had been reported up to 1897. Among them were 40 carcinomata and 31 sarcomata. His tabulation of these cases is given below. He describes briefly the cases reported from 1866. The synopses of these cases are as follows:

Buquoy[21] described a case in 1866. The patient was a woman, aged 24, who died suddenly 15 days after admission to the hospital. At the autopsy the heart was found to be involved in a large tumor mass, so that the myocardium was almost entirely destroyed. The tumor had extended to, and penetrated, the wall of the left ventricle and projected into its cavity. On the mitral valves were fresh warty excrescences. This tumor was reported as a primary carcinoma of the heart, but this is not certain, for no microscopical examinations were made.

Wyss[22], in 1866, reported a case of a woman who died from severe marasmus six months after admission. The autopsy showed a flaky new formation on the epicardium of the right ventricle. The right auricle was encroached upon by a hemispherical tumor 3 x 4 cm. x 20 mm. arising from the posterior wall of the auricle, extending into the atrium. It had grown

somewhat into the right auricle. The primary growth was in the mediastinum.

Ebstein,[23] in 1866, had a patient, aged 58, with a carcinoma in the esophagus. In the musculature of the left ventricle was a nodule the size of a walnut.

Prudhomme,[24] in 1867, reported a tumor of the heart which he thought was primary, but no microscopic examination was made.

Morgan,[25] in 1868, reported a primary carcinoma of the thyroid. There were nodules in the myocardium and pericardium. Carcinomatous thrombi were found in the heart.

Lober,[26] in 1875, reported a carcinoma of the testicle, with secondary growths in the bladder and kidneys. There was a nodule in the right ventricle at the septum. An embolus, containing carcinoma cells, was present in the pulmonary artery.

Besides the cases described by Boether, there are records of other cases which are briefly mentioned here. Fletcher,[27] in 1850, described a case with many secondary deposits in the heart and left pleural cavity. Wilks[28] found secondary tumor masses in the base of the heart, in 1854. Sibley,[29] in 1857, reported a case of encephaloid carcinoma of the right lung, which involved the heart. Moxon,[30] in 1867, found a secondary carcinoma of the heart in a case of carcinoma of the thyroid gland. Da Costa,[31] in 1880, reported a case of secondary carcinoma of the heart resulting in cerebral embolism. Martinotti,[32] in 1886, described a carcinoma of the heart with three metastatic nodules in the pericardium and four in the endocardium. Pic and Bret,[5] in 1891, described a case, a woman 67 years of age, who had a carcinoma of the esophagus, involving the lower third. In the heart there was a large carcinomatous nodule, situated on the pericardial surface of the left ventricle. There were also numerous nodules in other parts of the heart. In another case, reported at the same time, the patient had stenosis of the esophagus for six months before death. At autopsy a carcinoma, 10 cm. long, was found occluding the esophagus and adherent to the trachea. An effort to separate the tissues produced a tear in the esophageal tunic. The heart showed a metastatic nodule on the pericardial surface of the left ventricle, and metastases were present also in the liver, spleen and pancreas. Hektoen,[33] in 1893, reported a case of medullary carcinoma of the breast in a woman aged 50. The tumor had formed metastases in the lung, liver, Fallopian

tubes, uterus and pericardium. In the wall of the right ventricle, at the right border, just beneath the pericardium and projecting into the wall of the auricle, was a mass measuring 4 x 3 x 2 cm. It was firm, grayish white and infiltrating, and surrounded by blood vessels. The endocardium was not changed. Manifestly, the metastasis occurred through the coronary arteries. Rolleston,[34] in 1891, reported a case of epithelioma of the penis in which there was a metastasis in the apex of the heart.

Pelrourand and Molliere,[35] in 1894, described a secondary carcinoma of the heart. Loisney,[36] in 1895, described a carcinoma of the heart secondary to cancer of the liver. The patient was a man, age 67. There were no symptoms in the heart. At autopsy most of the liver was involved in the growth, and the central part degenerated and cystic. There were metastases in the right kidney and mesenetry. The heart was large, and the coronary arteries were partly calcified. On opening the left ventricle it was found to be much dilated. On the endocardium, extending into the auricle, was a moderately firm, pale yellow nodule, the size of a hazel nut. It was situated on the wall of the auricle, 1 cm. above the tricuspid valve. On microscopic examination the tumor in the heart was found to be adenocarcinoma. The muscle fibres of the heart were disassociated and destroyed by the growth of the epithelial cells. Bureau,[37] in 1897, reported a case of carcinoma of the esophagus, in which the growth had involved the pericardium and was adherent to the left auricle. The growth was the size of a pea and situated just beneath the endocardium. There was a stenosing growth of the esophagus, with ulceration and perforation into the middle lobe of the right lung. The sputum was fetid. There was a pleural effusion of 500 cc. The muscle cells of the heart were atrophied and infiltrated with cancer cells. Geipel,[38] in 1904, described a hypernephroma with a secondary tumor mass in the right auricle. In the right heart was a tumor, measuring 5.5 cc. long 32 mm. broad and 17 mm. wide, one-third lying in the auricle, two-thirds in the atrioventriculi ostium, resting on the tricuspid valve and between the chordæ. In the anterior wall of the left ventricle was a tumor nodule as large as a cherry. The hypernephroma was as large as two fists, growing into the renal vein. In the pulmonary artery was a tumor mass, measuring 20 x 7 mm., extending from

BOETHER'S TABLE OF CARDIAC CARCINOMATA OCCURRING UP TO 1897.

Author.	Kind of Tumor.	Position of Tumor.	Primary or Second.	Age and Sex.	Symptoms.
1. Andral (B*)	C med	r vent	primary.	35 m	angina pectoris
2. Lochner (B)	C fibr	r vent	"	47 m	hyp r v
3. Prudhomme (V-H†)	Carcin.	heart	"	?	?
4. Rulliere (B)	C fib.	myoc.	secondary	35 m.	h. hyper & angina
5. Velpeau (B)	C.	entire myoc.	"	?	?
6. Cruveilhier (B)	C.	r. atrium	"	34 m.	?
7. Hewett (B)	C. med.	r. au. & at. r. v.	"	40 m.	l. pleuritis & peric
8. Hewett (B)	C. med.	l. at. & vent mit. valv	"	59 f.	none.
9. Hewett (B)	C.	r. vent.	"	48 m.	syst. mur. at ap.
10. Cohn (B)	C. med.	entire myoc.	"	30 m.	none.
11. Günzberg (B)	C. med.	l. & r. vent.	"	40 m.	h. hypert and hydrothorax.
12. Holmes (B)	C. med.	r. vent.	"	23 m.	ang. pect.
13. Förster (B)	C.	r. v.	"	49 m.	dyspnea.
14. Paget (B)	C.	r. v.	"	58 m.	?
15. Heimpel (B)	C. med.	l. v.	"	56 f.	none.
16. Legallois (B)	C.	r. & l. v.	"	?	?
17. Bauchet (B)	C.	r. v.	"	?	?
18. Sims (B)	C. med.	r. & l. vt.	"	?	?
19. Ormerod (B)	C.	r. v.	"	?	?
20. Emmet (B)	C.	heart	"	?	?
21. Francis (B)	C.	l. v.	"	50 m.	?
22. Kundrat (W‡)	C.	aort. v.	"	70 f.	?
23. Ebstein (V-H)	C. epith	l. v.	"	58 m.	none.
24. Morgan (V-H)	C.	per. & end. cavities.	"	?	?
25. Lober (V-H)	C.	r. v.	"	48 m.	?
26. Bricheteau (S§)	C.	heart	"	24 f.	?
27. Sutton (S)	C.	heart	"	53 f.	?
28. Bayle (B)	C.	l. & r. at.	?	? m.	none.
29. Laennec (B)	C. med.	r. & l. v.	?	?	?
30. Trelat (B)	C. fibr.	l. & r. at.	?	?	?
31. Ollivier (B)	C.	r. v.	?	56 f.	none.
32. Billard (B)	C. fib.	entire myo.	?	3 child	?
33. Renauldin (B)	C.	r. v.	?	?	?
34. Segalas (B)	C.	heart	?	11 child	?
35. Sims (B)	C.	r. at.	?	?	?
36. King (B)	C. med.	base of h.	?	?	murmur.
37. Pagan (B)	C.	aort. v.	?	?	?
38. Williglk (W)	C. med.	tric. v. & con. art. pulm.	?	48 f.	vitium cordis.
			?	24 f.	?
39. Buquoy (V-H)	C.	r. & l. v. r. au.	?	24 f.	?
40. Wyss (V-H)	C.	r. & l. at.	?	59 f.	?

* B = Bodenheimer, Dissertation.
† V-H = Virschow-Hirsch, Jahresbericht.
‡ W = Wiener Klin. Wochenschrift, 1889.
§ S = Schmidt's Jahrbücher, B. 158.

the right heart, and in the lung was a mass as large as a hazelnut. Apparently, all the metastases were from the tumor mass in the renal vein.

Siegel-Delval and Marie[39] described a carcinoma of the esophagus extending to the cardia. It was ulcerated at the cardia, adherent to the liver and the lung. The pericardial fluid contained some fibrin flakes, but the pericardium was not adherent. In the inferior portion of anterior surface of the left ventricle was a sharply circumscribed neoplasm, situated in one coronary artery. It was surrounded by a zone of congestion and evidently was an embolus.

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THE ETIOLOGY OF EYE-STRAIN.

BY FRANCIS VALK, M.D.
Professor of Diseases of the Eye, New York Post-Graduate Medical School and Hospital.

To the general physician, the cause of eye-strain is usually attributed to some error of refraction, and probably, in the vast majority of our cases, this may be true; but it has been the past experience of the writer and of many others, that, after the refractive condition has been corrected—as far as our present knowledge will permit—our patients still present the same, or possibly somewhat modified symptoms. If we grant this proposition, which my own experience has proved to be true, then there must be some other causes that produce the symptoms of eye-strain than that of the refractive condition of the dioptric media.

Pohlman, in a discussion of the phylogeny and otogeny of the eyes of animals and of man, states that cases of eye-strain may be divided into two general clases[1], "those due to irregularites in refraction, and those due to muscular imbalance."[1] Gould, however, omits the latter condition, muscular imbalance, in his teachings and states that all cases of eye-strain are due to refractive errors alone; but, whereas he maintains that the correction of these errors, under an examination with a midriatic will relieve the ocular strain in all cases, yet he admits that he has many failures, which he refers to many other causes than that of a muscular imbalance. In a symposium on eye-strain, at Boston,[2] Myles Standish accepts both propositions with the assertion that epileptiform convulsions have been absolutely cured by the correction of the errors of refraction or the muscular imbalance; Greenwood inclines to the refraction theory only, and Kilburn to refractive errors with vertical imbalance, causing scoliosis and torticollis. Other speakers, at the same meeting, expressed similar views. Roosa would consider all eye-strain as due to some refractive error, but the writer must take the other side of the question. While I am willing to acknowledge that the vast majority of all cases of eye-strain are due simply to an

1. *Jour. Am. Med. Association*, May 6, 1905.
2. *Boston Medical and Surgical Journal*, Feb. 23, 1905.

existing error of refraction, yet certain cases also point to the fact that the symptoms may be due to a want of balance in the external ocular muscles without any objective or subjective evidence of a refractive error. The following cases will illustrate this condition:

Case 3953.—Mrs. A. L. B., age 25. Eye-strain, V. = 20/15. each. Rejects all glasses. Refraction almost emmetropic: Latent convergent squint, 10°. All asthenopic symptoms relieved by prisms.

Case 4190.—Mr. D., age 25. Eye-strain, V. = 20/15. Refraction emmetropic, no astigmatism. Rejects all glasses. Latent convergent squint, esophoria. Prisms of no service; all asthenopic symptoms relieved by shortening of the externus.

This presents two very similar cases, in one of which we have perfect relief with the use of prisms worn constantly, and in the other we could not get relief until the muscular balance was restored by an operation.

Case 4744.—Mrs. M. W. N., age 25. Worn glasses without relief; eye-strain and asthenopia. V. = 20/15, w + 1. D. cyl. ax. 90°, each eye. Latent convergent squint or esophoria with dextrophoria. Complete correction of the astigmatism with glasses gives complete relief.

Here we present the relief of eye-strain by correction of the refraction alone.

These three cases illustrate very clearly the usual means or method of treatment of eye-strain, in which the final result cannot be predicted until these means are tried; as correction of the refraction may fail, prisms may be tried without benefit and finally a suitable operation may be performed. Under this method of procedure, success will come in many cases, and our failures to relieve eye-strain will be very few.

But to proceed in the further consideration of the etiology of eye-strain, I cannot agree with Pohlman "that the human eye is subject to abnormality because of the domestication of man" and "unnatural environment," but rather would say that the supposed advance of civilization, which, in the present argument consists of an unnatural use of the eyes, is not the cause of the abnormality of vision, but that the abnormality is shown by the constant use. In other words, the eyes and the entire visual apparatus are the same today as in the aboriginal form, the high-

est form of evolution; but the advance of civilization demands such use of the visual apparatus that the cases of abnormal development must seek some artificial aid if they will, or must, continue to have the same strain placed upon them. The vast majority of our people do not wear glasses until they become presbyopic, and yet they can use the eyes without regard to time or place without any asthenopic symptoms, because they have a practically normal refractive condition and a normal muscular balance. I do not believe that phylogeny can prove that a refractive error or a muscular imbalance is due to environment or to civilization, but contend that astigmatism, refractive myopia, hyperopia and heterophoria are always anatomical rather than evolutional, while axial myopia, I consider as practically to be a pathological condition of the eyes.

To apply the theory of muscular imbalance to the evolution of the species seems to me a "far cry," as the divergence of the axes of the orbits bears, in my opinion, no relation to the axes of the eye itself; and Hansen Grut's theory of the position of rest has no foundation of fact, according to my own investigations.[3] We may make the same statement as above in reference to the refractive condition of the eyes, as I do not think that the presence of an astigmatism of any kind can be due to the "present civilized state," nor can I agree with the proposition, "the more perfect our eyes are, the less are they adapted to the present conditions." The emmetrope with normal muscular balance does not suffer from eye-strain, a fact well proved from an examination of the records of our opthalmological offices.

We may well ask the question, then, why is eye-strain so prevalent at the present time? Now, we may say that our present civilization has been the cause of so much eye-strain, if by civilization we mean the constant use of the eyes for near work. The demand for reading matter has increased a thousand fold during the past two decades. Our country is dotted all over with the circulating libraries lending out books to "old men and maidens, young men and children," whereby they spend their time reading, instead of indulging in that healthy relaxation and exercise that the system needs.

If we glance at the matter in this way, the etiology of eye-strain becomes very apparent, as, it goes without argument,

3. Valk on Strabismus, p. 51.

that, if the muscles which control our seeing function are constantly in action and we have an abnormal condition in the ocular apparatus, then nature must rebel, and we have the phenomena of eye-strain as the result. Perhaps we may say that the emmetropic and orthophoric eye will never feel the effects of any strain on the seeing function. These eyes will stand an immense amount of work, as reading, even during an exhaustive sickness in some cases; but, while I believe that the majority of all people have this emmetropic eye, or perhaps slightly hyperopic, yet a large number do have certain refractive errors or a muscular imbalance—perhaps associated with some constitutional conditions—that must feel the effects of eye-strain if they attempt to use the eyes with the same requirements that may be placed on the emmetropic eye. We may, then, consider that the essential causes of eye-strain are, primarily, a refractive error which may be a simple hyperopia, a myopia, or an astigmatism due to an unequal refraction in the two principal meridians of the cornea, and secondarily, a want of balance in the straight muscles of the eyes which will give an inequality in the ocular motility.

It would be a long story to relate the many symptoms, as stated to us in the consultation room by our patients, and from which they seem to suffer more or less. I might say that many of these symptoms are heard in the stories of all patients of a physician; but it seems to me that some of them have a special reference to the ocular apparatus, and these must call for a special examination. Among the latter we find backwardness in children, inattention at school, rubbing, blinking and pressing the lids, blurring of the vision, tilting or holding the head forward or backward, headpain caused by reading, writing, and sightseeing, vertigo and dizzy sensations, and headpain around, or back of the neck and around, and in the eyes. All these symptoms noted evidently refer or point to some abnormal condition of the eyes, either refractive or muscular, and, unless there is some other obvious cause for the symptoms, call for a careful examination of the ocular condition. This examination is imperative before we try to relieve certain symptoms with drugs that may be due to an abnormal condition of the eyes. Headache, that most common expression of the present day, has been referred to the eyes by the laity, but this is only a symptom

that may be due to many other conditions; yet it is the one most commonly related in the office of the oculist. As a corallary to this paper I would refer the reader to a recent paper by Coggeshall and Maccoy[4] on this subject, in which they take the position that "eye-strain headache is due, not only to a refractive error but may be due to a muscular imbalance;" and on the subject of migraine, these writers state that an individual with migraine, and having a decided neuropathic diathesis, will probably have an error of refraction or a muscular imbalance as the exciting cause of the phenomena of migraine. At the same time we must admit that other conditions of auto-intoxication may be the underlying cause of the pain.

It goes without argument that the diagnosis of eye-strain must be along certain lines that necessarily require certain special training. It is also, without doubt, a part of the work of a physician, a part of his armatarium for the relief from the ills that flesh is heir to; and if the demands of a so called civilization require more use of the eyes than in the good old days we like to talk about, then these demands must result in certain symptoms, be they functional or not, which come within the practice of medicine as much as the mechanical fittings of a truss or a brace for an orthopedic deformity. An optician may be qualified to fit certain refractive cases and may do it perfectly, but he is not qualified to do all the work that is required for the correction of all cases of eye-strain; while the law requires and demands that all conditions of the eyes that may arise do come within the domain of the practice of medicine. I do not believe that the eye and its appendages are any different today from what they were fifty years ago; our population has increased an hundred fold, our opportunities to use the eyes at the near point have enormously increased, and our boys and girls are constantly using the eyes reading the many books and papers so freely and cheaply supplied. The result is that we must meet this condition by all the means at our command, when the annatomical condition of the individual requires our aid. The mere fitting of glasses is not the *sine qua non* of the treatment of eye-strain. Many other conditions must be taken into our consideration, and indeed the intelligent practitioner will realize that the correction of a refractive error, or that of an imbalance of the

4. *Journal Am. Med. Association*, January 4, 1908.

eyes, may be of vast importance in the successful treatment of certain of his cases. Perhaps this statement might raise the question in our minds, should all physicians be oculists? By no means. If civilization has advanced in the past two or three decades, so has opthalmology; so much so, that the subject has become too extensive for a physician to master all its details, even those of eye-strain, and yet be a good general practitioner. But while the physician in our larger cities may refer his cases, the urban physician cannot readily do so, and he should be able to understand the subject of the refraction and the motility of the eyes. Let him know at least what may be considered as the standard of vision and motility of the eyes, as well as the normal sounds of the heart and the percussion note of normal lung tissue, and then, having recognized an abnormal condition of the eyes, he may or may not decide to correct it. Is there a normal condition of the eyes? That question is too broad and scientific to answer in a paper of this scope, but we may make some suggestions in relation to it. To do so, I will not consider all the intricate methods of diagnosis used at the present day—we must leave these cases to the ophthalmologist, who is more or less proficient in the use of certain scientific instruments—but every physician can have a test card displayed in his office for the purpose of testing the visual acuity. Furthermore, he may have a candle placed at twenty feet in front of his patient, and with a small set of prisms or even the Rod of Maddox, he can test the motility of the eyes. Now, with this simple outfit, and bearing in mind that we do have a standard of vision and of motility, he can readily decide the presence of an abnormal condition that may be the cause of an eye-strain and of its functional symptoms. Let me give that standard in a few words. Our standard vision is usually designated as 20/20., which implies that the eyes are adjusted for rays of light coming from infinity or twenty feet, and that the dioptric apparatus will focus these rays perfectly on the retina with the eye at rest. But our people are so familiar with the size and shape of the letters that I find we must raise this standard somewhat, and I think that 20/15. would be nearer the normal standard; that is to say, we should see letters at twenty feet whose secondary axial rays form a smaller angle than one of five minutes or to make this subject more simple, we can see letters at twenty

feet that should be seen only at fifteen feet. To repeat, if each eye will see letters at twenty feet that are marked for fifteen feet, then we may say that the eye has normal vision. It is true that many eyes with abnormal refraction may have normal vision, but that subject comes within the domain of the specialist and not that of the general physician. Our object is to call the attention to, and to point out, the method of examination where the vision is blow the normal, as if the record only shows V. = 20/40., then the sight is subnormal, and we may have some evidence of the cause of the eye-strain. Having now examined the visual power, we now proceed to test the motility of the eyes, or their power to fuse images, by the prism test. First, place a red glass before one eye, and as the patient looks at the candle, note if he has double vision and if so whether the images are crossed or homonymous; that is to say, is the red glass on the same side on which the red light is seen, or is it on the opposite side? If on the same side, it indicates a possible paresis or at least, a weakness of one of the abductors or externi. If on the opposite side or crossed, it indicates a possible paresis or weakness of one of the adductors or interni. The Maddox rod is used in the same way and shows the same conditions. We follow this test by removing the red glass and place a prism of six degrees over one eye with the base of the prism placed inwards. If this does not cause any diplopia, then remove that prism and place a prism of twenty degrees over the eye with the base outward. Again if we have no diplopia, we may use stronger prisms in the same way. Finding the strongest prism, base in or out, with which the patient can fuse or blend the images of the candle at twenty feet, if we then discover that the degrees of these prisms bear the relation of one of abduction to three or four of adduction, it is very doubtful if we have any muscular imbalance. Such is the simple, yet excellent test for the standard of the motility of the eyes. These are tests that should be in use in every physician's office, and the routine use of them in all cases that present any symptoms of eye-strain may assist in the diagnosis. To carry our examination beyond this point would come within the work of a specialist, who is more fully competent to work out the possible latent conditions, as astigmatism and certain muscular errors. These simple tests were the first ones that I was taught in my early work in ophthal-

mology, and, in my experience since that time, they hold as good to-day as three decades ago. In presenting the above statements of the correct vision and motility, it must be understood that in some cases we may have eye-strain with the most perfect vision, and it has been suggested, as an axiom, that "the better the vision, the more the reflex troubles." While I doubt this, yet we do find that many slight errors of refraction are the prime cause of certain reflex symptoms. It is doubtful if any line of treatment will be successful if these cases are due to a refractive error, or to a muscular imbalance. These people must submit to a careful scientific examination of the eyes for the correction of the existing abnormality by glasses, or such other methods as may seem necessary. This examination is essential so that we may eliminate eye-strain as the cause of certain other reflex symptoms. But even accepting the proposition that eye-strain may be the cause of the symptoms, yet, with their accurately fitted glasses, even under a midriatic, such persons will still feel the strain on the accommodation with pain about the eyes. If so, then we may find indicated certain general tonics, as restricting the use of the eyes, outdoor exercise, and, as a local application, I have found very useful a solution of Eserine sulphate, 1/40 gr. to the ounce, one drop in each eye three times a day. This drug has a tendency to stimulate the ciliary muscle to action, and so lessen the strain on the accommodation in reading. The use of Eserine as a ciliary tonic was suggested by the late Dr. C. R. Agnew at the time he was surgeon of the Manhattan Eye and Ear Hospital, and it has been of much service to me in many cases of eye strain after the full correction of the refractive and the muscular errors. Furthermore, as regards any internal treatment in these cases of eye-strain that may be due to any muscular imbalance, Seguin, in 1892, published a very interesting article in the *New York Medical Journal* on eye-strain, particularly in reference to an original weakness of the nerves of adduction and abduction, in other words, any lateral muscular imbalance. In his final conclusions as to treatment in cases where the symptoms are increased by an act of accommodation and by convergence, or, we may say, where there is a tendency for the eyes to roll outward when at rest, exophoria, Seguin recommends the use of nux vomica or strychnia, and claims they are sometimes cured by a course of this drug. Furthermore, he says, if we find the

fault in the power of abduction, or a tendency for the eyes to roll inward, esophoria, then the use of cannabis indica, bromides and atropine for the complete rest of the accommodation, is indicated. I think this line of treatment will hold good at the present time. These are all cases of muscular imbalance, or, as our honored president, Roosa, would call it, latent strabismus, and may be corrected by drugs, as Seguin suggests, by wearing prisms or by an operation if needed. Change of scene and travel are useful but should not be ordered until we have fully corrected all the visual defects. Again, after all these things have been done, we may find it necessary to allow the patients to use the eyes only by degrees, by the method called Dyers Plan—which I have found useful in some cases—in which we allow them to use the eyes only for stated periods certain times a day. In closing this paper, we must all admit that eye-strain does exist in far greater numbers than in former years, that it is due to a civilization that demands much more constant use of the eyes, that many of our people must have certain abnormal development of the visual apparatus as regards the refraction and the motility, and that the general physician should have, or be aware of, some means by which he may realize the possible presence of eye-strain and then be able to take such means for its correction as may seem best.

IDIOCY AND THE ALLIED MENTAL DEFICIENCIES IN EARLY CHILDHOOD.

BY HERMAN B. SHEFFIELD, M.D.
Instructor in Diseases of Children, New York Post-Graduate Medical School and Hospital

In the majority of instances idiocy is congenital, and due to ante-natal disease or arrest of development of the fetal nervous system. Some mental deficiencies of infancy are the result of traumatism before, during and after birth. A still smaller number of cases of mental degeneracy is the product of disease or defective development in early childhood. The stigmata of degeneration, therefore, are in full bloom in the first few years of life, and can readily be demonstrated by careful examination of the patient.

In our effort to arrive at a correct conclusion, it is essential to bear in mind the unreliability of parents' views as regards the mental condition of their children—most parents look upon their offspring as the wisest in the land—and the fact that no very sharp lines of demarcation can always be drawn between infants with normal intelligence and those below the average. Hence, the importance of minute analysis of the family history of the patient; the past history of the patient, particularly as regards nerve disorders and traumatism; and the physical and mental conditions of the child since birth, especially as to their progressive or regressive character.

A nervous heredity being traceable in the great majority of cases of feeble-mindedness, *the family history* is often very enlightening in obscure conditions. Grave neuro-psychopathic affections, especially if occurring in the immediate family, demand careful consideration, and a history of dipsomania or syphilis in the parents should always be looked upon with suspicion. Inquiry should be made about the condition of the mother during pregnancy. It has not rarely been demonstrated that infants conceived during convalescence of the mother from prolonged attacks of exhausting diseases (*e.g.*, typhoid) were born idiots. Furthermore, serious domestic troubles, mental anxiety, and physical distress, extreme fright and violent traumatism in the mother may so disturb the normal evolution

(Sheffield) FIG. 1.—Hydrocephalic idiot.

FIG. 2.—Microcephalic idiot.

of the fetal organism as to create grave central disturbances in the offspring. Notice should be taken also of the fact that certain types of mental degeneracy are peculiar to certain races of humanity—often without any discernible cause—as, for example, amaurotic family idiocy in the Hebrew race.

The association of feeble-mindedness with any of the aforementioned predisposing factors cannot invariably be accepted as proving the hereditary pathogenesis of the case in question. On the contrary, occasionally totally insane parents beget perfectly sane children, and *vice versa*. Anticipation of an herdeitary predisposition, therefore, should not deter us from careful scrutiny of the patient's *personal history*, particularly as pertaining to traumatism sustained during birth (compression or fracture of the skull during tardy or instrumental delivery, etc.) or after, and the diseases the child suffered from up to the time of examination.

No one morbid manifestation in the *past history* of the patient is as corroborative of the latter's abnormal mentality as the occurrence of repeated attacks of convulsions during its early development. Whatever the cause—be it meningitis, gastrointestinal intoxication, uremia, exanthematous disease or trauma—the very fact that severe convulsions occurred justifies the assumption of some pathologic alteration in the central nervous system, sufficiently grave to predispose to mental impairment. Whenever possible, an attempt should always be made to trace the exact origin of the mental deficiency. Too much stress, however, should not be placed upon the information received. For, at best, histories are only guesswork, unless furnished by very intelligent sources—rather rarely to be expected when dealing with degenerates.

Having obtained all details as to the patient's family and personal histories, our next, most important, work should be to determine the apparent *physical* and *mental conditions* of the child, especially with the view of comparing them with those of a normally developed child of the same age.

The *head* of the idiotic child presents several characteristic physical stigmata of degeneration. The cranium of a hydrocephalic (see Fig. 1) is large, ball-shaped, and its circumference widest at the temples. It contrasts strongly with the small, delicate face. The fontanelles are separated, the eye-

brows are scarcely indicated, and the mouth and nose are small. In microcephalus (see Fig. 2) the head is small, narrow anteriorly and devoid of posterior projection. It greatly resembles that of an animal. The fontanelles are prematurely ossified. The eyes are small, the ears project, and the nose and the lower jaw are large. In the syphilitic (see Fig. 3) the head is unevenly enlarged. The skull is irregularly bossed and traversed by prominent blue veins. The nose is small and often saddle-shaped. The eyes are large and bulging. The lips are usually thick and the angles of the mouth not rarely marked by rhagades. The cranium of the Mongolian (see Fig. 4) idiot is somewhat smaller than normal, rounded, with the occiput running quite parallel with the plane of the face. The face is sunken, the nose short and broad and bound laterally towards the eyes by distinct vertical folds. The cheek bones are prominent, and the tongue protrudes. No characteristic physical signs of degeneration are apparent on the cranium of the amaurotic idiot (see Fig. 5), except that owing to the general atony of the musculature, the patient is unable to hold up his head. The face is delicate. The skull of the cretin (see Fig. 6) is rather larger than normal, sparsely covered by thin, lustreless hair, and set upon a thick, short neck. The face is weak and senile, the eyelids and lips are thick, the tongue is heavy and often protrudes from the half closed mouth. Meningitic, encephalitic, or paralytic idiocy (see Fig. 7) usually presents no characteristic cranial physical signs, except when due to severe traumatism at birth or after. In some, congenital cases there is marked flattening of the temporal bone of one side corresponding with the lesion in the brain (porencephaly, etc.). In infantilism (see Fig. 8) the skull is smaller than normal, and the face is either plump and senile (typus Brissaud), or thin, delicate and infantile (typus Lorain).

The teeth of the great majority of idiotic children are irregularly implanted, faulty in form, and excessive or deficient in number. Owing to irregularity of the dental arches and size of the teeth, the patient is frequently unable to close his mouth —which should not be mistaken for the open mouth associated with nasal obstruction—and the constantly dribbling saliva not rarely leads to painful excoriation of the chin. The lips are often congenitally malformed. The palate is high and narrow

FIG. 3.—Syphilitic idiot.

(Sheffield)

FIG. 4.—Mongolian idiot.

FIG. 5.—Amaurotic idiot (diostasis recti abdominis).

(Sheffield)

FIG. 6.—Cretinic idiot.

and quite frequently clefted. Internal inspection of the nose usually reveals numerous deviations from the normal construction. The ears often project or are asymmetrical. Aside from the prominence of the eyeballs spoken of, idiots often present diverse anomalies varying from simple errors of refraction to total absence of the eyes.

Except the presence of pseudolipomatous masses in the myxodematous, there are no pathognomonic physical peculiarities of the *trunk* which may be helpful in the differential diagnosis between the numerous forms of mental backwardness. Most idiots are undersized, present more or less marked deformities of the thorax and spine, large abdomen, hernias and narrow pelves. The genitalia are often undeveloped and malformed.

The condition of the *extremities* varies in the different types of idiocy. The hydrocephile often suffers from paraplegia with spastic rigidity of the muscles, and is thus unable either to walk or stand. The upper extremities are usually normal, and only occasionally affected by contractures and athetotic movements. The microcephalic idiot is an extremely restless creature and rarely sits or stands still even if supported. Some few of them are completely rigid, and others, when they grow older, manage to walk about. The syphilitic, as a rule, is helpless, principally as a result of deformities of the extremities and rarefying and softening inflammatory processes at the articulations. The child walks as if paralyzed, if it can walk at all. The Mongolian idiot begins to walk at a much later age than the normal baby. His joints are weak, and his hands and feet plump. Amaurotic idiocy is characterized by muscular atony which gradually terminates in general paralysis. The cretin has a peculiar dragging and awkward gait, and often presents deformities of the extremities and thickening of the joints. Unilateral or bilateral hemiplegia with contracture and athetosis is pathognomonic of the paralytic idiot. Where one hemisphere is involved he has a tottering gait. In infantilism the extremities are apparently normal in shape, but their musculature is weak and flabby. A radiogram (see Figs. 9 and 10) of the hand usually reveals backward development of the centers of ossification of the carpal bones and of the epiphyses of the metacarpals and phalanges.

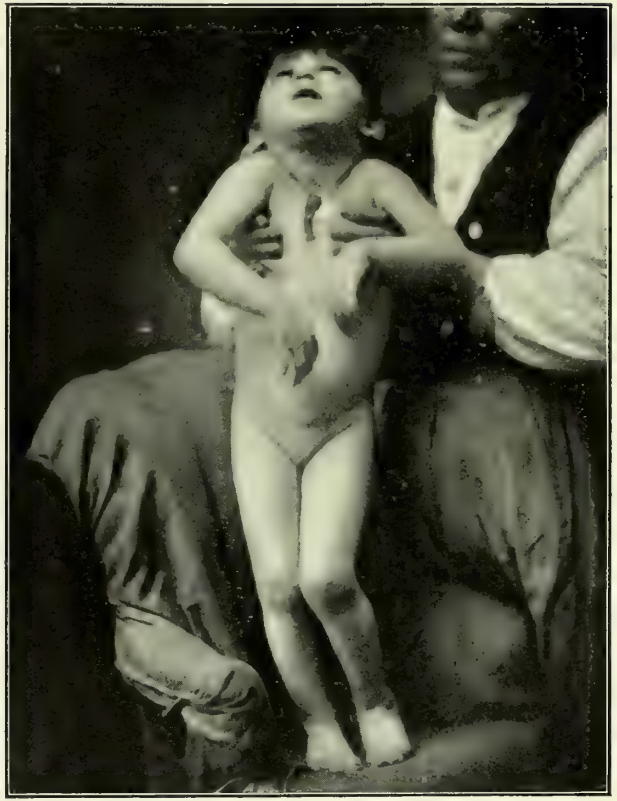
In addition to the aforementioned stigmata of degeneration

idiots often present asymmetry, malformation, superabundance or deficiency of fingers and toes (see Fig. 8), club-foot, ankyloses, dislocations of the extremities, diastases (see diastasis recti abdominis, Fig. 5), and as a result of lesions in the brain and spinal cord, divers forms of paralyses and contractures, and local and general atrophies, etc.

As infants at best are not endowed with extraordinary mental powers it is in them quite difficult to distinguish normal from abnormal mentality. Still, with experience one learns to depict the *mental signs of degeneracy*, especially by bearing in mind the progressive character of the normal infantile mind.

The mental faculties of an infant become appreciative with the evolution of the senses of hearing and sight. A perfectly normal baby almost fully controls these senses by the end of the third month. As it grows a few months older it begins to show signs of power of attention, perception and memory—it is delighted by bright objects, recognizes familiar faces, more or less appreciates pain or pleasure, etc. When nine months old it is usually capable of understanding certain words spoken to, and manifests the tendency to imitate sounds and syllables. At about one year of age, if properly trained, most infants are in full control of the voluntary musculature—creeping, standing, and sometimes walking—and partially so of some of the body functions, such as defecation and urination. Premature infants and those of feeble vitality from other causes may acquire some of these faculties at a much later period of time, and yet grow up to be normal intellectually. However, while no standard time limit can be set down for the establishment of one or the other mental function, failure of their manifestation at an age considerably beyond the supposed normal period, *e.g.*, failure of a child nine months old to indicate a fair possession of the power of attention, or to recognize persons who are in its constant attendance, ought to always arouse suspicion as to the normal mentality of the child in question.

In idiots or mentally backward children the power of *attention* is either very poorly developed, or entirely absent. They fail to take notice of their surroundings, stare vaguely into empty space, or move the eyes irregularly in all directions, apparently seeing nothing. This want of attention may be due to partial or total blindness, as is frequently the case in amaurotic family



(Sheffield) FIG. 7.—Paralytic idiot.

Fig. 8.—Infantilism (deficiency of thumbs).

idiocy, of central origin without involvement of the eyeball. Occasionally the patient may be subject to congenital cataract, microphthalmos, coloboma iridis, irideremia, lesions of the vitreous, strabismus, and similar visual defects which in conjunction with other stigmata facilitate the diagnosis.

Except in encephalitic or amaurotic family idiocy, defective *hearing* of central origin is rather uncommon. On the other hand, deafness is not rarely observed as a result of congenital malformation or acquired disease of the auditory canal. In the latter event a history of normal hearing at an earlier age is usually obtainable.

Genuine idiocy is invariably associated with a voracious appetite, and owing to imperfect development of the senses of *taste and smell*, no choice is manifested as to the kind of food given. Everything that comes along is rapidly devoured; hence the frequency of gastrointestinal disturbances in feeble-minded children. The sense of smell is sometimes so obtuse that even irritating odors are not productive of local or reflex phenomena in the respiratory tract. On the other hand, occasionally idiots, like animals, are endowed with a hyperacute sense of smell.

Most idiots are *insensible* to touch, pain, heat or cold—anomalies of sensibility which explain the frequency with which such children are subjected to external injuries and voluntary bodily mutilations. In some of them, however, especially in those with marked defective vision, tactile sense is so highly developed that by this means alone they are able to recognize persons who feed and care for them.

Congenital deafness is, of course, associated with mutism. But even where hearing is intact, few idiots are able to speak. Some of them, by imitation, do learn to utter a few words, but their expressions usually bear no relation to any distinct desire or action, and they understand words spoken to them no better than what they speak. Moreover, their power of *imitation* is very much delayed in development or may never become manifest—all depending upon their faculty of attention. In partial idiocy, such as mild forms of infantilism, cretinism, encephalitis, or microcephalus, the power of *conversation* may reach a certain degree of potency, but their vocabulary is usually very limited and fragmentary.

This form of *pseudo*-deaf-mutism, like genuine deaf-mutism, is not necessarily correlative with the degree of intellectual development of the idiotic children in question. Some idiots, *e.g.*, microcephalics, may incoherently chatter for hours, and yet be no wiser than those who never utter a single word, since the intellectual capacity of the idiot depends upon the extent and location of the lesion in the brain.

With few exceptions, idiots are unable to acquire, retain, associate or evolve ideas, reason, judge, or appreciate their personality, their actions or their surroundings. Instinctively they may cling to those who feed and take care of them, like animals obey their masters, after prolonged training perform certain actions, and even manifest a certain degree of reasoning power. In the majority of instances, however, their performances are mechanical and automatic. They may for hours lie or sit in one position and indulge in certain movements, without by attitude or expression indicating the desire for a change, or even betraying any discomfort previous to or after the acts of defecation or urination.

As compared with the different varieties of idiocy, *intelligence* is least allotted to the amaurotic idiot. Deprived of sight and hearing since early infancy, limp and languid as a result of the ever increasing atony of its musculature, the helpless creature gradually loses all its senses, and, fortunately, also its life. Microcephiles are nearly as badly endowed with intelligence as the former group, but their stupidity is not progressive in character. As they grow older they are able to feed themselves, and with hearing intact they may learn to talk—talk without sequence or measure. Genuine microcephalic idiots are obstinate, vulgar, and brutal. Almost the exact opposite characteristics are observed in hydrocephalic idiots. They are ordinarily soft, gentle, timid, sorrowful, but little impressionable or curious. As very marked cases of hydrocephalus usually succumb at birth or soon after, those surviving usually possess a greater degree of intelligence than microcephiles, nay, at times they may grow up to be perfectly normal. The mental impairment following meningitis, or encephalitis, varies with the extent of the brain lesion produced by the inflammatory process. Deaf-mutism, aphasias, and amaurosis being common sequelæ, little intellectual capacity can be expected. In the

Fig. 9.—Normal wrist of child 6½ years old.

Fig. 10.—Wrist of idiot 10 years old. (Sheffield)

absence of these defects, the little patients may gradually acquire a fair measure of intelligence. The same observations practically hold good for syphilitic idiocy. Tics and convulsions are not rare in both of these types of idiocy. The Mongolian idiot is a restless creature. Totally idiotic in early infancy, he gradually shows signs of improving mentality. He learns to appreciate his surroundings, and to make himself understood by a language of his own. He learns to run about at an earlier age than most other idiots, and not rarely shows destructive tendencies. Under suitable treatment cretins often attain a fair measure of intellectual development. Like hydrocephiles they are timid, gentle, and unassuming. They retain their childish tastes for a long time, and sometimes for life, if left untreated. In the latter respects infantilism differs little from cretinism. In infantilism, however, there is much greater control of the muscular system, and quicker response to medication and training.

Occasionally mixed forms of idiocy, genetous idocy (Fig. 11) are encountered which do not quite correspond to any of the types enumerated, demanding for their correct interpretation greater care and scrutiny and more elaborate methods of examination. Limitation of space, however, forbids further dilation upon the vast and complex subject in question.

CONTRIBUTION TO THE SURGERY OF ACUTE PANCREATITIS.

BY FRANZ TOREK, M.D.
Adjunct Professor of Surgery, New York Post-Graduate Medical School.

Previous to the present decade the diagnosis of pancreatitis was rather rarely made except at autopsies. Since the time that the disease has been treated surgically, it has been recognized and described much more frequently, and at the present time physicians are beginning to become aware of the fact that pancreatitis is not so very rare. If we fail to diagnosticate an ailment, the reason most frequently is that it does not occur to us that there is such a disease. If it were generally appreciated that, when a patient is suddenly attacked with severe pain in the upper part of the abdomen, we must think of acute pancreatitis as one of the many possibilities, the disease would doubtlessly be recognized more frequently. The symptoms, however, are sometimes so urgent that there is no time to exhaust the means at our disposal to establish the diagnosis; we may often get no further than to say to ourselves that there is a severe affliction in the upper part of the abdomen, which requires prompt surgical interference. Delay in such a case, simply to gain time necessary for making a probable diagnosis, would not be in the interest of the patient.

In this article I shall report three cases operated upon in 1907. One of these died, the other two recovered. In two the diagnosis was not made previous to the operation; in the third, pancreatitis was considered to be very probable.

Case I.—W. H., male, age 24 years; admitted to the German Hospital, May 20, 1907. Alcoholic history. Has had two previous attacks of abdominal pain, one six weeks ago, lasting one day, and one five weeks ago, lasting two days. Two days before admission he was taken sick with sharp, severe pain in the epigastrium, and he vomited. Later, the pain became more severe in the lower abdomen, but not especially over McBurney's point. Small movement of the bowels this morning.

On examination we found a small-framed, fairly well nourished man, with a drawn facial expression, looking very sick. Pupils reacted to light and distance. Cervical lymph nodes enlarged. At the bases of the lungs flatness on percussion; no friction sounds. Heart negative. Liver

a little higher than normal. Abdomen distended, tender all over, especially in the hypogastrium, more on the left side, than on the right. Flatness in the flanks. Tremor of the hands; occasional motions like those in tetany. Pulse about 100, regular and full. Temperature between 100 and 101° F. Blood examination: White blood corpuscles, 22,000; polynuclear cells, 91%.

The patient was taken to the operating room within two hours after his admission. The symptoms pointing to diffuse peritonitis, a long median incision was made. Free turbid fluid escaped, which was seropurulent toward the pelvis. The appendix was found normal. Small spots of whitish material were seen here and there on the peritoneum and the intestines. These could not be rubbed off; they were areas of fat necrosis. These were distributed throughout the peritoneum, very sparsely on the small intestines; a little more on the descending colon; still more, but still in discreet spots, on the mesentery of the small intestine; considerably more and confluent areas on the mesentery of the ascending and descending colon; very thickly on the mesentery of the transverse colon and in its neighborhood, particularly at the pancreas, which was enlarged and firmly united with the stomach, duodenum, omentum, and liver; the whole conglomerate of organs being covered by this necrotic tissue. Lavage with saline solution; closure of wound without drainage.

After the operation the patient was quite restless; he slept at short intervals, talked aloud while apparently sleeping, and picked at the bedclothes. Saline enemata, one quart, containing an ounce of whiskey were given on the first and second days by the slow drop method according to Murphy. On the second day delirium set in, gradually getting worse. Patient got out of bed. On the third day the delirium became more and more violent; it was viewed as delirium tremens and the patient was isolated. The temperature, which hitherto had gone no higher than 101° F., rose in the night of the third day to 102.8° F., and on the fourth day, just before death, to 103.4° F.

The autopsy, performed by Dr. F. C. Wood, revealed the presence of red hepatization in the greater part of the right lung and deep congestion of the posterior part of the left lung. In the abdomen no fluid was found, nor other sign of peritonitis. The findings in respect to fat necrosis were the same as those at the operation, except that in the autopsy report no mention is made of small, discreet spots on the intestines, such as were found at the operation. It seems, therefore, that these were absorbed in the interval between the operation and the autopsy. The pancreas was found to be about two and one-half times the normal size and of firm consistency. No hemorrhage; no pus. On section, white granular areas of irregular form and varying size were seen. The duct of Wirsung was patent and contained no bile; its opening into the duodenum was separate from that of the common bile duct; No stones in the pancreatic or biliary ducts. Liver normal; biliary system normal; ducts patent. Stomach normal; intestine normal, except for follicular enteritis in the lower part of the ileum. Anatomical diagnosis: Acute pancreatitis; lobar pneumonia.

Looking at the case critically, we find very little indeed that could have helped us to make the diagnosis of pancreatitis. The only symptom that might have directed attention to pancreatitis, was to be found in the patient's history, that the attack had begun with a sharp pain in the epigastrium. On the other hand, there were enough signs to mislead. The great tenderness over the entire abdomen, which was most marked in the hypogastric region, pointed rather to diffuse suppurative peritonitis of appendical origin. The fact that the tenderness was greater on the left side than on the right, did not militate against that assumption, as it is not rare to find that the more recently involved parts of the peritoneum are the most sensitive. The pain and tenderness, therefore, as they presented themselves on the patient's admission to the hospital, could not help us to make the diagnosis of pancreatitis. The extreme acuteness of the symptoms did not warrant any extensive laboratory studies. The high leucocyte count, and especially the very high percentage of polynuclear cells, indicated that operation was urgent. The autopsy showed that the pancreatitis was not secondary to diseases of the biliary system. The case also shows that even the most acute cases with extensive fat necrosis do not necessarily die on the first or second day of their disease, as has been claimed. This patient died on the fourth day after his operation and had been sick for two days before he was operated upon. Furthermore, his death was due to delirium tremens and lobar pneumonia. Without these complications he would doubtlessly have lived longer. In fact, as certain spots of fat necrosis found at the operation were not seen at autopsy, there is reason to believe that, without complications setting in, it may be possible for a patient to recover, the necrotic areas being reabsorbed. Mayo Robson observed a case of extensive fat necrosis that recovered. Formerly it was held that acute pancreatitis was regularly accompanied either by hemorrhage or suppuration; but cases have been reported in which neither of the two were present, and this case belongs in that category.

Case II.—L. B., male, age 34 years; admitted to the medical service September 3, 1907. Heavy drinker. Five months ago he had an attack of pain in the upper part of the abdomen similar to his present attack. Eight days ago he was taken sick with cramps rapidly increasing in severity, followed by flatulence and vomiting and severe frontal headache.

The pain in his abdomen started in the left hypochondrium, then extended to the right. The lower abdomen was not much affected. The vomitus was small in amount and slimy, but not bloody. The urine was high-colored; the frequency of micturition increased. He complained of heart-burn and backache; also of insomnia. No jaundice. His chief complaint was cramps and vomiting. On examination his conjunctivæ were slightly yellow. Heart sounds faint, no murmurs. Abdomen, general tenderness, especially in the upper left quadrant; no masses, no rigidity. Liver dulness from fourth space to one finger's breadth above the umbilicus, where its border could be palpated. It was very tender.

On September 9th he was transferred to the surgical department. In the right upper quadrant of the abdomen a hard, very tender mass was felt, with ill-defined borders, but giving the impression of being the gall-bladder. It did not extend beyond the nipple line, but the tenderness extended farther out. The mass extended almost to the umbilicus. Intestines appeared to overlie it. The abdomen was distended and tympanitic; no free fluid could be made out. Temperature 104.7° F.; pulse, 132. Blood examination: Leucocytes, 11,400; polynuclear cells, 75%. The impression was that of a perforative peritonitis from some organ in the right upper quadrant, and of the different organs the gall bladder was considered the most probable. An incision was made parallel to the right costal border. The gall-bladder, liver, stomach and adjacent intestines were all matted together. The adhesions were separated and the gall-bladder carefully examined, but, except congestion shared by all the organs in that region, nothing abnormal was found The stomach and duodenum also presented no perforation. Finally, pus was reached somewhere near the pylorus, but behind it and the duodenum, welling up in large quantities through the posterior parietal peritoneum at a point corresponding to the head of the pancreas. The pus was thin and foul, with clumps like rice in it. The rest of the peritoneum had been previously walled off, so it was kept clean. Drainage-tubes and gauze were inserted and the wound partly closed. A culture from the pus showed the presence of colon bacilli.

The temperature declined gradually after the operation until, on the fifth day, it reached 100° F., and remained for a long time between 100° and 101° F. with occasional rises due to retention; it declined again as soon as the retention was relieved. Recovery was rather slow, the discharge continuing for a long time, although it showed a gradual diminution. On November 24, a selected diet was given, to the practical exclusion of the starches; after this the discharge seemed to diminish more rapidly. The urine was examined frequently. It almost always contained a trace of albumin and hyaline and granular casts up to the last few weeks of the patient's stay in the hospital, when it was negative. He was discharged cured on Jan. 25, 1908, and was presented by me to the matriculates of the Post-Graduate Medical School on Feb. 1, 1908.

It will be seen that, when this patient was referred to the surgeon, the symptoms were such that an exact diagnosis could not

be made. The most that one could say was, that there was an acute process in the upper abdomen urgently requiring surgical interference. While the symptoms tallied with those of acute pancreatitis, they corresponded equally well to perforation of the gall-bladder, stomach or duodenum, or to rupture of an appendix situated well up under the liver.

Case III.—M. L., male; age 18 years; admitted Sept. 24, 1907. No alcoholic or venereal history. He had typhoid and measles in childhood. Had a number of injuries. He fell from a tree four years ago, hurting his spine; recovered after eight weeks' stay at a hospital. Also broke a clavicle. Bowels generally regular. Onset of present illness twenty-four days ago while in Kingston, Jamaica. He complained of constipation and of pain in the left part of his abdomen, where there was also tenderness; the pain was increased by motion. From the time he left Jamaica until he reached Colon, he had no movement of the bowels for five days. During that time he had general abdominal pain which was later localized in the left upper part of the abdomen. His pain became worse after eating solid or semi-solid food. At Colon he was in a hospital for eight days. While his pain was great throughout, it was much aggravated when his bowels moved after taking medicine. Since leaving Colon he has had pain only in the upper left part of the abdomen. His constipation required much medicine. There has been no blood in the stools. He had chills and fever in the morning of the day of his admission and had also had them several times before.

On examination it was evident that he was in pain. He belched gas. Tongue heavily furred. Heart and lungs normal. Abdomen tense; markedly rigid and tender over the left side. Marked pain also in the left lumbar region and that of the left lower ribs. Percussion of the left upper part of the abdomen dull. The patient prefers a quiet posture. Temperature 102.8° F., soon rising to 103.2° F.; pulse, 96, of good volume. Blood examination: Leucocytes, 25,800; polynuclear cells, 81½%; no plasmodia. Examination of stomach contents after test-meal revealed presence of five cu. cm. of undigested food, consisting of starch and fat, also some mucus; otherwise negative. Feces, examined on Sept. 26, contained no blood, few leucocytes, some mucus, some starch, a little fat, some vegetable fibres, and bacteria. On that day the temperature had gone down to normal, but the pain persisted; so an examination under narcosis was made. The examination showed the presence of a tumor mass in the left upper part of the abdomen. Inflation of the colon proved the colon tympany to merge into the tumor as palpated; but it was dull at that point. Its course was not deflected medially by the tumor. The tumor itself did not move on respiration, and was almost immovable. The diagnosis of a retro-peritoneal tumor was made, and, as the examination of the urine was negative, it was believed probable that it represented the tail of the pancreas. A blood examination, on Sept. 27, showed a practically normal condition. On Sept. 28, an operation was performed. Although the tumor was recognized as retro-peritoneal, an abdominal in-

cision was made, as it gave better opportunity for exact observation and diagnosis. Left rectus incision, six inches long. The omentum, lying over the colon covering the mass, was found adherent to the posterior peritoneum. A long tag of omentum passed down and around the gut to become attached to the mass underneath, which proves to be the much enlarged tail of the pancreas, acutely inflamed. This condition doubtlessly explained the obstinate constipation of which he complained. The kidney lay high up and was of normal size. The patient was now turned upon his right side and a left posterior incision made, similar to that employed for nephrotomy. The dissection was carried into the depth below and to the medial side of the kidney. Blunt dissection was very much facilitated and hastened by bimanual work with the left hand in the peritoneal cavity. On reaching the pancreas, an aspirating needle was inserted with negative result. A long forceps was then introduced into the organ and its branches opened, but without disclosing the presence of pus. The diagnosis of acute pancreatitis without pus was then made. The abdominal wound was closed; the posterior wound was left open and drained with tube and gauze.

On the second day after operation, the temperature rose again to 102.4° F. A change of dressing revealed a wound discharge which, while not profuse, was rather ill-smelling and distinctly purulent. Staphylococcus aurens was found in the pus. It appears, therefore, that there was a focus of pus in the pancreas which was not opened at the operation, but found its way out later. The temperature went down on the following day. The wound condition remained about the same for two or three days. Then the discharge gradually diminished and the wound closed, the patient being completely cured.

In this case the symptoms were not so stormy as in the other two, except that the pain was intense. The presence of a palpable tumor in the left hypochondrium was a great help in the diagnosis. Its retroperitoneal character was made out by inflating the colon and observing its position in relation to the tumor. It was most probable, therefore, that the tumor was pancreas, although the negative urine report would not exclude the possibility of a kidney tumor. On exploratory laparotomy, the manner in which the omentum was wrapped about the pancreas at once gave proof of the inflammatory nature of the tumor. The way in which it also bound down the colon, gave an interesting illustration of the manner in which obstinate constipation and even intestinal obstruction may occur as a consequence of pancreatitis. The great pain which he suffered may have been, in part, due to pressure of the enlarged organ on the ganglia of the solar plexus. Drainage through a posterior incision, in cases where the pus has not yet broken through

the peritoneum, is doubtlessly the correct procedure. In Case II, where the peritoneal cavity was already invaded, a posterior incision was scarcely indicated.

Reviewing these cases and comparing them with those observed by others, the following remarks suggest themselves. As regards the etiology, the observation has often been made that alcoholism is a cause of acute pancreatitis. Now, while the first two cases here reported had a most pronounced alcoholic history and thus support that statement, my third case did not take any alcoholic drinks. Disease of the biliary system is another cause of pancreatitis. In my first two cases the gall-bladder and ducts were examined at the operation, the first one also with particular care at the autopsy, and were found to be normal. In the third case no examination of the gall-bladder was made, the incision being on the left side; but, as there was no indication whatever in the history and symptoms pointing to disease of the biliary apparatus, I may be free to say that in my cases this etiologic factor was inoperative. As regards diseases of the stomach and intestine as a cause of pancreatitis, the autopsy in the first case revealed a follicular enteritis in the lower ileum; in the second case the history pointed to the presence of gastroenteritis. It will be remembered that both of these were alcoholic patients. In the third case, however, there was nothing pointing to a previous gastroenteritis, the bowel trouble being fully explained by the mechanical obstacle presented by the attachment of the omentum.

The urine was not tested for Cammidge's pancreatic reaction in any of these cases. This test is doubtlessly valuable, but it is quite laborious and takes a good deal of time, especially that part of it which requires the repeated slow cooling of the flasks with the least possible disturbance. It also presupposes a fair degree of experience to ensure against errors. In Cases I and II there would have been no time to make the test before operation, even if pancreatitis had been suspected; in Case III there was time enough, and the failure to make the test must be admitted as neglect.

In many cases of pancreatitis, a tendency to hemorrhage has been observed, and calcium chloride has been given to counteract that tendency. In none of my cases was any such

medication administered; yet there was practically no hemorrhage at the operation, nor was there any subsequently.

In order to recognize diseases of the pancreas more frequently than we have heretofore, the most important thing, after all, is not to forget that organ, when we encounter troubles in the upper abdomen. This being borne in mind, a thorough study of the physical signs and metabolic changes, as well as chemical tests, will often help us to establish the diagnosis.

MASTOIDEOTYMPANOTOMY: THE OPERATION OF ELECTION FOR PERSISTENT OTORRHEA AND ACUTE MASTOIDEO-TYMPANIC OSTITIS.

BY W. SOHIER BRYANT, M.D.
Adjunct Professor of Diseases of the Ear, New York Post-Graduate Medical School and Hospital.

A form of mastoid operation, which I have called mastoideotympanotomy, has given me most satisfactory results in those cases in which otorrhea is persistent and in acute ostitis with involvement of the tympanic walls. I wish to advocate this technique in preference to any other, because it fulfils more completely the requirements for a greater number of cases. In all but the rare instances of extensive tympanic caries, our operation is able to entirely relieve the pathological condition. In the writer's experience, tympanic caries sufficiently extensive to counter indicate mastoideotympanotomy is found only in a fraction of 1% of all cases seeking, through a mastoid operation, a cure of the above conditions.

The advantages of the author's operation are threefold. It is not very extensive; it does not jeopardize the life of the patient; it does not require close curettage of the outer wall of the labyrinth. This curettage sometimes appears to cause infection of the labyrinth and to give rise to serious intracranial complications in the Schwartze-Stake operation. Our operation, on the other hand, is of comparatively short duration and not only retains as much sound conducting function of the tympanic contents as still remains at the time of operation, but even in most cases markedly increases the auditory capacity. The disadvantages of our operation are that it is not applicable to every single case without exception, as we have previously noted, and that it requires careful bone dissection.

According to our technique, the operator makes any of the usual aural incisions and removes as much bone from the mastoid process and cells as his judgment deems necessary in the individual case. The author thinks it is wise to be conservative and take no risks by leaving bone which may later interfere with the repair of the wound.

The posterior wall of the external osseous auditory canal is

removed exactly as is done in the ordinary Schwartze-Stake radical mastoid operation or tympanectomy, with this difference that in my operation the annulus tympanicus is carefully dissected and left in position. All the adjoining walls of the mastoid antrum are removed. The outer walls of the aditus ad antrum and epitympanum are removed as completely as possible without injuring the peripheral attachment of the drum membrane, the posterior ligaments of the incus and the suspensory ligaments of

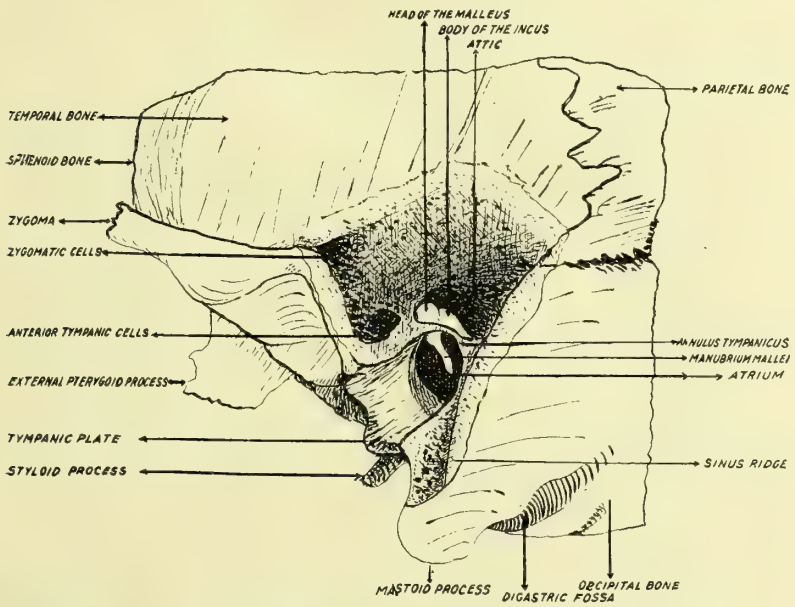


FIG. 1.—Outline of temporal bone showing the field of a mastoideotympanotomy.

the malleus. Finally, as much of the hanging wall of the osseous meatus is removed as is possible without uncovering the dura mater of the middle cranial fossa, and all the zygomatic cells present are thrown open. It is important to insure complete drainage of the atrium by extensive section of the drum membrane or cicatrix that may choke it. Finally, a plastic meatal flap should be cut. To accomplish this the dermoperiosteum is incised, beginning at the inner end of the lower wall of the external meatus just external to the attachment of the drum mem-

brane. The incision is carried outward and downward completely through the soft tissue till it reaches the concha. At this point the knife is swung backward and then upward far enough to make a meatus sufficiently large to admit the index finger. The free angle of this plastic flap is sutured upward and backward to the posterior edge of the periosteum above and behind the bone excavation, and the cat gut is drawn tight enough to widely distend the meatal orifice. The mastoid wound is closed by three or four deep periosteal sutures and a continuous subcutaneous suture. Pack the meatus lightly with iodoform gauze, taking care that a little of it shall penetrate to the bottom of the antral or post-tympanic bone excavation. A wet dressing should be applied. After twenty-four or forty-eight hours the packing is removed from the meatus and not replaced. When the tympanic membrane has closed, the tympanum is inflated through the Eustachian tube in order to aid the normal readjustment of the sound conducting mechanism.

The following instruments are used by the author in the operation: the rongeurs, curettes, front-bent gouge, scalpel, artery clamps, bistoury, forceps, scissors, retractors, needle-holder and needles.

The following cases are reported to show the fact that even when the convalescence is comparatively prolonged the results are far superior to those obtained by other methods.

Case I.—A woman 50 fifty years old. Operation performed at the New York Post-Graduate Hospital. Previous history: Purulent discharge from both ears for many years. When I first saw the patient she had had for five weeks, symptoms of mastoiditis on the right side, edema, tenderness and temperature ranging up to 101° F. She had not slept for many nights on account of pain in the head. On examination, I found she had tenderness of the tip of the left mastoid. The right mastoid was tender low down and behind; no tenderness of the neck in front. Temperature, 101°+F. The mastoiditis had been the cause of her developing septic depressent insanity, and she looked forward to a speedy death.

The patient was prepared for immediate mastoid operation on the right side. I made a T-shaped cut and found the dura deciduous on the face of the mastoid. The bone bled freely; the bleeding was annoying throughout the operation. I en-

tered the bone by first removing the tip of the process. The cellular structure was softened and filled with bleeding granulations. The softened bone extended well over the base internal to the digastric fossa. The inner table was softened and it was necessary to uncover the sigmoid sinus throughout its whole length. An epidural abscess covered the vertical limb of the sinus. The sinus appeared unaffected. The dura mater covering the sinus and extending some distance behind it was thickened and red, thus showing pachymeningitis. I uncovered a very large area of the dura. The intracranial tension appeared to me normal. Large cells were opened far back in the occipital bone. Some pus was found under pressure in the cells lying

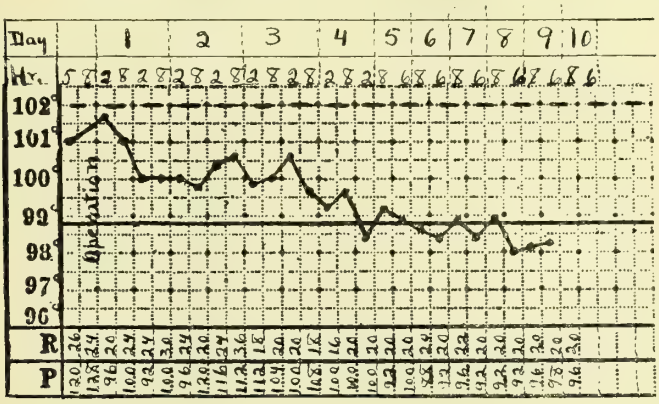


CHART I.

posteriorly and internally at the base of the skull. The posterior meatal wall was removed nearly to the attachment of the drum membrane and the epitympanum was exposed. I closed the wound around two small cigarette drains, one extending into the antrum and one to the jugular bulb, packed the meatus lightly and applied a wet dressing. The operation lasted one hour and fifteen minutes; much delay being caused by the constant bleeding.

Owing to the patient's mental condition, convalescence was very slow. She tried to take her own life by jumping out of the window. She deranged the dressings several times and pulled them off twice. Hair became matted in the wound which was partly open, resulting in the infection of the wound. No packing

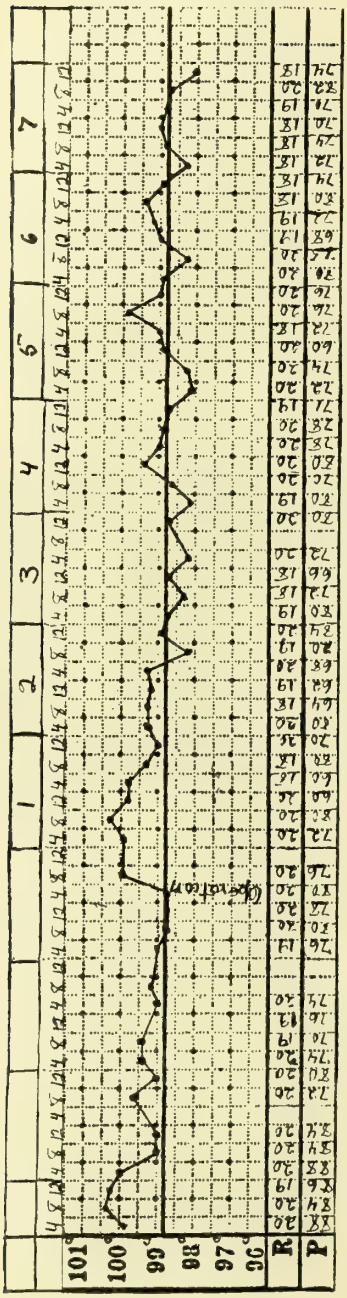


CHART II.

was used. A thin slough formed over the surface of the wound. No granulations appeared for a long time.

On the 13th day of the convalescence the patient's mental condition had much improved. The slough was detaching from the wound and granulations were appearing. On the 17th day the middle ear was dry. On the 35th day of convalescence the canal, drum membrane and mastoid wound were healed. Watch heard at two inches from the right ear. On the 67th day the left ear had healed after cleansing treatment and the watch was heard at 10 inches; on the operated side the watch was heard at 4 inches. On the 109th day the patient said she heard best with the right ear. The watch was heard in this ear at eight inches. Post aural scar very slight, with an even surface. The perforation in the drum membrane had closed. On the 111th day tympanic suppuration recurred in the right ear. The suppuration in the right ear again ceased two days later. On the 135th day of convalescence the watch was heard at 25 inches in the right ear; in the left ear at 11 inches. The post aural scar had very much diminished and scarcely showed.

Case II.—Patient a man 65 years old. He had no ear history previous to this attack. It began 12 days before the operation with headache, dizziness, nausea and discharge from the ear. The ear condition seemed to the patient to have been brought on by an attack of grippe. At the time of operation there was extreme tenderness over and behind the mastoid tip. There was also slight tenderness over the mastoid antrum. Profuse discharge of foul pus. Tympanic membrane bulged superiorly. I made the usual crescentic post-aural incision and opened the bone by removal of the mastoid tip. I found an extensive cellular area involved and filled with granulations. There was no pus except in the zygomatic cells and cells at the base internal to the digastric fossa. The softened bone required an extensive dissection, but I did not uncover the dura. I removed the posterior wall of the osseous meatus clear up to the annulus and opened the tympanum from above and externally. I closed the external wound with deep and superficial sutures and applied a wet saline dressing.

On the seventh day of convalescence the posterior wound had healed solid by first intention.

On the 18th day the perforation of the drum membrane had

closed. On the 32nd day the watch was heard at two inches. The meatal sinus was epidermatized and firmly healed. On the 52nd day the watch was heard in the right ear at 26 inches; in the left ear at 14 inches. On the 60th day the patient " caught cold in the head." There was a relapse of suppuration in the right tympanum and spontaneous reopening of the drum. Two days later the right tympanic membrane had healed again. The hearing was poor and there was a little discharge from the meatal sinus. On the 86th day the meatal sinus had again healed. On the 152nd day the watch was heard in the right ear at 28 inches. The scar was barely perceptible.

The shortest period of complete convalescence with plus normal hearing which I have had after this operation, was eleven days.

Conclusion.—From the point of view of the patient's life, comfort and hearing, mastoideotympanotomy is the operation of election for chronic middle ear suppuration and acute ostitis involving the tympanic walls.

THE POST-GRADUATE STUDENT.

BY H. BEAMAN DOUGLASS. M D.
Professor of Diseases of the Nose and Throat, New York Post-Graduate Medical School and Hospital.

Education does not end with school life. For a while all go along on the same path, both sexes in childhood having much the same needs and experiences. After a time differentiation begins. Some accumulate knowledge in a practical way; others, under the guidance of teachers, acquire a prolonged technical education which is used as a foundation upon which to build again.

However prolonged the special student life may be, it eventually ceases. The individual under the delight and glamor of graduation exercises, baccalaureate sermons and the congratulations and felicitations of his friends, is at length thrown upon his own resources and now shapes the accumulated material and new experiences into the structure which he calls his life.

Student life, however, is not confined to the college walls. The term "self-made" man proves that those who have not had the advantages of early education and prolonged instruction under the guidance of teachers have, nevertheless, directed themselves so well and accumulated an experience resulting in a life so aggressive and successful, that it almost makes one gasp to think what the self-made man might have been had he enjoyed the privileges, advantages, and general culture which are the best fruits of the university life.

During his university career the student acquires a mass of information, much of which, like the scaffolding employed in the construction of a building, is subsequently discarded. Nevertheless, it serves a necessary purpose. Such an educational course enables a man to choose the quicker and easier path to his goal, rather than to struggle forward over an uncleared and unmapped way.

At last those who are to develop the self-made life, and those who have had the advantages of education, meet on common

ground. All the subsequent experience of both must be in the nature of an experimental life, if progress is to be made. One sees careers checked by dry rot; spoiled by inability to expand; ruined entirely by a disinclination to attempt the untried way, or wasted from lack of independence. This furnishes a reason for the common belief that the best men in actual life are not those who were the best students in the Universities. The men who shone brilliantly during their college career are frequently so dependent upon others that they cannot develop independently and self-reliantly. After a brilliant and rapid rise and explosion, like a rocket, they drop suddenly and darkly to earth.

The goal of success may be reached under various standards. Opinions vary as to what constitutes success. I think it may be taken for granted that success in life means progress, development, consecration to work, concentration of purpose, the pursuit of ideals, and the accumulation of a competency sufficient to enable one to furnish himself with the proper environment and afford the means of providing for himself and family.

The pursuit of these aims in proper proportion constitutes a well-balanced life. Each individual, however, emphasizes for himself one or more of them which absorbs his main energies and determines the bent of his career. After pursuing for a while one of these ideals, it frequently happens that the individual changes his purpose, accepts another, pursues this also for a while, then changes for another. Thus his interests are dissipated, his life appears frivolous and purposeless, and his accomplishments mediocre. He who selects one or possibly two of these purposes, and pursues them with tenacity, determination and result, may justly be accused of being narrow-minded and restricted in his outlook. In middle age he also finds his life a failure; for, while it meets the approval of the public and is stamped with apparent success, he feels the need of a wider outlook and realizes that he has lost much of the beauty and pleasure of life.

The post-graduate student, as we see him in our college, manifests many admirable traits and some of these faults. He comes because he is dissatisfied with his progress; feels the need of growth and development; desires association and contact of

men in a wider and more active field, from whom he hopes to receive inspiration.

The problem how to instruct a class of such men is, indeed, a difficult one, owing to the wide variations in their mentality, personal experience and general qualifications. Some have finished their college life and take the post-graduate course as a substitute for hospital training. These minds are young, bright, and filled with theory. Others are staid and venerable practitioners, who have been removed from the college environment long enough to forget most of their technical knowledge. They have acquired a mass of experience the value of which has never been fully utilized, because they have been isolated from medical centres and almost entirely dependent upon their own resources. Such men are hungry for definite information; have clear ideas of what they wish to gain, and, having obtained it, are in position to use it at once and to advantage. Another student in the same class will represent the unsuccessful man. He has tried this and that and failed in all. He has been farmer, storekeeper, justice, and finally selects medicine as a field in which he hopes to find success. He has wasted his energies and lost his ideals. All his views are tinged with commercialism. Next to him sits a man who has used all his opportunities. He is educated in the special line in which he seeks additional knowledge, and is often as well equipped as the man from whom instruction is sought. This one is simply broadening his field of observation.

With a mixed class of this kind, it is almost impossible for the instructor to present his subject in such a manner as to interest and hold the attention of all. Therefore, the primary essentials of post-graduate teaching are small classes and the close and personal contact of pupil and instructor.

The importance of teaching in small classes has been realized by the undergraduate medical schools and by most of the Universities. Most classes are now split into smaller bodies which come directly under the personal supervision of the instructor or professor. Especially is this true in Princeton University, where a plan is being developed which brings each student in close contact with a tutor, and every possible stimulus is given to original thought and work. This need of personal instruction and influence in post-graduate work cannot

be too persistently emphasized by our governing bodies. I believe it is the only way in which the requirements of the post-graduate student can be met. I would welcome a curriculum which would abolish long clinical lectures, or even the demonstration of cases in which the student cannot participate actively in the examination.

All post-graduate work should require the student to come in close contact with the patient, in order to learn all the methods of examination and practice, and to carry out the simpler forms of treatment and perform the minor operations. The work of the instructor should be merely supplementary. He should guide, aid, suggest, direct, and furnish stimulation and inspiration. It is only by such methods that the graduate student can be properly fitted for his work.

The theoretical presentation of a subject can be easily obtained by reading. This can be done alone. Many of our post-graduate students are already well versed in the literature of the subject in which they are seeking objective knowledge. Laboratory work is one of the special branches which attracts the post-graduate student, and in which he often does the best work. It would be distinctly to the advantage of our school if the laboratory method of instruction could be adopted in clinical work.

Besides the need of practical work, our students also feel the necessity for drawing fresh inspiration from the teacher. This may be given by carefully demonstrating methods of investigation; giving him the opportunity to see and perform operations; making the course suggestive for his future development by indicating lines on which he may make observations after leaving the school, or even by suggesting original clinical or research work which may be carried out during his course.

It is a deplorable fact that such an outline as this would not meet with the approbation of all the students. Unfortunately, some of them are more interested in watching operations or copying prescriptions written on the blackboard than in doing the work which would qualify them to perform these operations or to use the prescriptions with intelligence.

Effective post-graduate teaching demands the highest ability in the teacher. It gives the student something else than that which he obtained in his undergraduate course; for it not only

presents fresh knowledge, but also furnishes moral support and inspiration for better work in the future. Therefore, the post-graduate teacher should be a priest in medicine; he should be progressive and still maintain a high degree of practical conservatism; energetic, but never a faddist; critical, with the power of balancing methods and differentiating their value. Without being didactic, he should be able to present his subject in a practical and interesting manner and, by his personality and skill, to quicken the interest and ambition of the student.

Post-graduate instructors in this country have almost invariably studied in the clinics of Europe. Even to-day this field is very attractive to the post-graduate student. On this side of the water we have a language which is understood by all the students. Even the finer distinctions of words and emphasis carry weight and meaning; the clinical material is abundant and interesting, and the equipment of the graduate institutions is first class.

It is only fair to acknowledge, however, that real post-graduate opportunities have developed in the United States only within the last quarter of a century. Before then Europe was the only place to obtain proper instruction. Then, as now, men received a healthy stimulus to future work from the association of well-known men. There is a certain glamor connected with the name of a famous man, and an enthusiasm absorbed from him which makes the work all the more interesting. It is also fascinating to wander through the various countries; to observe their institutions, manners, and men; and to feel the influence of centuries of history, architecture, and art. Without doubt, the pathological and laboratory work on the other side is superior to ours. We must never forget, however, that a serious difficulty exists in one's lack of familiarity with the languages. One is impressed every day with the importance of understanding the fine distinctions which can be conveyed only by emphasis and inflection. With an incomplete knowledge of a language, it is often difficult for the student to make progress. Often when he believes that he has completely grasped the meaning of the instructor, he finds that an imperfect understanding of the language has led him into error, and an entire revision of thought is necessary. Thus he is in constant mental turmoil and confusion.

Unquestionably, a man who is seeking post-graduate instruction may visit Europe to great advantage to increase his knowledge and experience of men, to compare methods of work and broaden his views generally; but his real, earnest, and enthusiastic work and development of technique can be done best in the post-graduate schools of the United States.

TRUE PRIMARY OVARIAN PREGNANCY; OPERATION; RECOVERY.*

Report of a Case with Microscopical Findings.

BY SIDNEY D. JACOBSON, M.D., M.R.C.S. (ENG.), L.R.C.P. (LOND.).

That " Nature hides her secrets well " is an old but true saying. It ought, therefore, not be wondered at that the Ancients, lacking proper training and without the more modern instruments of precision, knew very little of the finer (histological) processes involved in the advent and progress of pregnancy. It was at one time believed that, before impregnation could follow coitus, the male " *Liquor proliferus* " or " *prolificus*," must unite with a similar fluid of the female within the uterus. Obviously, such a view excluded the possibility of the occurrence of extra-uterine pregnancy. Nevertheless, it sometimes happened that such a condition was found at autopsy. It would have been un-orthodox and altogether reprehensible to admit that the teaching was faulty; so, in order that the fact might be made to fit the theory, the most absurd and fanciful superstition was invoked to furnish an explanation. Accordingly, it was declared that a miracle had occurred; others stated that this was a freak of nature; still others blamed the evil influence of witchcraft; while some, more radical in their views, dared to assert that it was surely a case of ruptured uterus.

In due course of time, reports of cases of extrauterine pregnancy accumulated to such an extent that it no longer sufficed to explain them away on such flimsy grounds—some more plausible theory had to be invented. It, therefore, happened that the medical teachers of that time originated the brilliant idea that impregnation took place if the male semen was, after coitus, absorbed into the circulation of the female, and afterwards distributed to the proper place. One of the beneficent results of this absurd teaching was, that a distinct step forward was attained, because at last it was admitted that physiology must be studied, to solve the problems involved; and henceforth

*From the Pathological Laboratory of the New York Post-Graduate Medical School and Hospital.

the pernicious habit of trying to explain physical phenomena by sophistry was abandoned. Another result of this new teaching was that extrauterine pregnancy having been, as it were, officially recognized as possible, a great number of cases were now observed and reported.

For scientific purposes, a classification of these cases soon became necessary. Not content with a simple definition, an attempt was made to classify extrauterine pregnancy as to its location, etc. This was, before long, amplified by the addition of subdivisions, so that eventually there arose a bewildering array of different kinds of gestation. To illustrate this point, I shall cite but a few examples taken from Gurgui, which will show how authors vied with each other to report something more wonderful than their predecessors. Thus, Noel, in 1779, described a case of *vaginal pregnancy* which was supposed to have been observed in 1765. Josephi and others spoke of pregnancy in the *urinary bladder*. Chavanne, in 1862, mentioned a case of pregnancy in the *cervix uteri*.

Perhaps the simplest classification was the one adopted by Boehmer, in 1752, who divided extrauterine pregnancy into tubal, ovarian, and abdominal. As to ovarian pregnancy—the one which interests us most—Bianchi and Boehmer subdivided this into the external and the internal varieties. Velpeau was a most determined antagonist of any theory which recognized the possibility of impregnation within the ovary. He said:* How can there be an internal ovarian pregnancy if, as is well known, the Graafian follicle must burst and the ovum be extruded before impregnation can take place? Other authors, like Thompson (1838), Pouchet (1842), Bischoff (1845), took this apparently reasonable view and stoutly defended it. In 1845, Mayer began a crusade against abdominal pregnancy in general and ovarian pregnancy in particular. Kiwisch successfully refuted Mayer's arguments.

The internal variety of ovarian pregnancy having been shown by Velpeau, Bischoff, Mayer and their followers to be impossible, Cauwenberghe, in his memorable prize essay, demolished the other variety, namely, the external. This was in 1867, and inaugurated the second important battle fought on this question. It seems that the decisive point as to whether an ovarian preg-

* *Op. cit.*, p. 213 (see Literature appended).

nancy was of the internal or the external variety depended upon its location—inside or outside of the tunica albuginea. It only remained for Waldeyer to prove that there is no tunica albuginea, and both parties were completely routed.

Now followed a time of confusion which was increased by the occasional battles royal among the medical leaders while discussing this question of extrauterine pregnancy. Innumerable cases of ovarian pregnancy were reported because the products of conception (?) were found within the ovary. Thus teeth, bone and hair of different lengths, were triumphantly exhibited as proof of such claim. They had not yet learned the significance of dermoid cysts. In 1845, Mayer wrote a startling essay in which he boldly denied the existence of ovarian pregnancy and proved that the so-called ovarian pregnancies recorded up to that time were something else. The result was, that since Mayer's time, authors became a little more careful in their statements, and the extravagant claims of enthusiasts dwindled to the vanishing point. It became apparent that, to establish a case, accurate data would be demanded. Furthermore, certain points or criteria would have to be met before one could sustain his claim of demonstrating a particular kind or variety of extrauterine pregnancy. Until the middle of the last century, these demands or criteria were lacking as regards ovarian pregnancy, so that it sufficed for a single sign (like absence of the ovary on the affected side) to stamp a doubtful tumor ovarian pregnancy. The logical outcome of this universal demand for scientific accuracy was a classical work by Cohnstein, who set up the following four criteria to prove ovarian pregnancy:

1. Absence of the ovary on the affected side.

2. Connection of the sac with the uterus by the thickened ovarian ligament.

3. Lammellation of the sac which must be lined with cylindrical epithelium. Direct connection between sac-wall and tunica albuginea.

4. Amniotic cavity in immediate connection with a Graafian follicle or a corpus luteum.

Criterion No. 3 was subsequently refuted as irrelevant by Spiegelberg, who substituted the very important one, that the Fallopian tube on that side must not in any way be implicated

in the growth. Spiegelberg's criteria of ovarian pregnancy are used to this day. They are as follows:

1. A tumor, evidently the ovary, occupying the place of the latter.
2. Connection with the uterus by the ovarian ligament.
3. Normal, non-adherent tube, on the affected side.
4. Definite ovarian tissue demonstrable in some part of the sac.

To the casual observer these four criteria of Spiegelberg might seem ample, but on close investigation it will be clear that these are not sufficient. For, if no fetus is found, it may be because it has become attached secondarily to some other intraabdominal organ, or if found connected with an ovarian cavity it might have become so accidentally or secondarily. In either case this would not constitute a true, primary ovarian pregnancy. It seems, therefore, that two more criteria should be added, namely, (*a*) the fetus must be found situated within the ovary; (*b*) organic connection between this fetus and the ovary must be demonstrated—to show that it actually grew there. It may be objected that a fetus can be found within the ovary only in an early stage of its development, because after it reaches a certain size it could no longer be retained within the organ. In answer one may say, that this is precisely what happens and is the reason why secondary abdominal pregnancies occur; also the intraligamentary kind. It is on account of the lack of room for further development that these primary ovarian pregnancies become secondarily abdominal or intraligamentary. Having ceased to be ovarian, they must be named something else. The word ovarian in this connection denotes merely the anatomical or topographical location of the gestation sac; and it matters not if a pregnancy, primarily ovarian, becomes in due course of time abdominal.

From a study of the subject it will appear that all pregnancies are primarily ovarian, in the sense that impregnation takes place ordinarily in the ovary. Perhaps it will serve a better purpose not to analyze the evidence in support of this statement here, as it would lead away from the subject under discussion. This matter will be taken up under its proper heading in the appendix to this paper.

To resume, then, it would appear that two more criteria should be added to those usually demanded, when determining the

question of ovarian pregnancy, making six points in all and reading as follows:

1. A tumor, evidently the ovary, and occupying the place of the latter.
2. Connected with the uterus by the ovarian ligament.
3. The tube on that side perfectly normal and non-adherent.
4. Definite ovarian tissue demonstrable in some part of the sac.
5. The fetus visible in a cavity of the ovary.
6. Demonstrable organic connection between the fetus and the ovary.

With these six points scientifically proved, no doubt need be entertained as to the nature of the case. With one or more of these lacking, none ought to be considered as proved.

For the sake of completeness it would, no doubt, be desirable to pass in review all cases of this nature recorded in the literature. This, however, is hardly feasible; firstly, because it would presuppose on part of the writer, mastery of many ancient and all the modern languages, and secondly, it would require unlimited time and opportunities for reading. Besides, an attempt at such a compilation would augment this paper to the dimensions of a volume. For the present purpose it may suffice to give a very brief résumé of the more likely cases of ovarian pregnancy, recorded as such or quoted as authentic by the more modern writers on the subject. It is assumed that the above detailed six criteria of ovarian pregnancy, are accepted as sufficient, and this " rule of six " will be applied impartially to all the cases reviewed, to test their claim to that title.

The first case of extrauterine pregnancy recorded in the literature is the one described by the Spanish-Arabian physician Albucasis, who died at the beginning of Hegira 500 (the 12th century 1106–'07 A.D.). In 1618, Riolan Jr., known as the chief anatomist of his day, described a case of tubal pregnancy observed by Mercer four years previously. The same author at the same time also mentioned two other cases, one by Marquard, about 1588, and one about 1608. Following these cases, further observations of tubal pregnancy were recorded by Riolan in the first half of the 17th century. Vesal reported a case in 1669, and this one was followed by a number of other instances recorded by various observers.

It is noteworthy that the anatomists and surgeons of that period received these reports with reserve, not to say suspicion.

In fact, the more conservative men were guarded in their expression when reporting such observations. Thus, Mauriceau claims but to recognize in his case a "hernia uteri," and Bartholin* states that Riolan did not believe in the cases he (himself) had published in book form, but had pretended to believe in them merely out of respect for the Court Physician, Pierre Seguin, who had observed them. About that time the study of the ovary received a strong impetus. The Graafian follicles were discovered and studied, and although at first misinterpreted to be "ovula," the labors of Harvey (1651), Nil Stenson (1667), Van Horne (1668), Kerckring (1671), Swammerdam (1672) and especially Regnier de Graaf, dispelled many doubts which had arisen in this connection. From this time on, the numerous cases of rupture of the uterus disappear from the literature and reports of extrauterine pregnancy take their place.

In his inaugural thesis Gurgui gave a beautiful exposition of the subject of ovarian pregnancy, and divided the cases into two parts: in the first part those reported up to 1845, and in the second those reported from 1845 to 1880. In the classification adopted in the present paper, Gurgui will be followed closely, many parts being a literal translation from his work; but, in addition, all the probable cases reported in the literature from 1880 to 1908, which have been accessible to the writer, will receive attention.

PART I.

All the cases of ovarian pregnancy reported before 1845.

Group I.

These are obviously dermoid cysts of the ovary mistaken for cases of ovarian pregnancy.

Division 1.—Characterized by the presence of hair in a sac within the ovary—19 cases.

Bauhin, John.....1578
Bauhin, Caspar.....1600
Fabrice de Hilden....1618
Wepfer.....1660
Tyson. 1681
Poirier, Du.....1700
Schamberg.....1703
Schacher... 1720
Haller.....1743
Menghini and Bonzi...1745
Castel.....1751
Chirac.....1766
Saxtorph.....1779
Warren.....1783
Weinholt.....1800
Merriman.....1812
Horn.....1814
Koehler..... ..1825
Cruveilhier.....1835–1842

* *Op. cit*, p. 260 (see appended Literature).

Division 2.—In which teeth, bone and sometimes hair were found within the ovary.

Lamzweerde.....1671
Blasius.....1675
Tyson, 3 cases.....1679
Stalpart.....1682
Needham.....1683
Sampson, Birch.....1683
Mulebancher.....1687
Teroude.....1690
Marci.....1690
Mery.....1695
Nichols.....1696
Ruysch, 3 cases.....1716
Grambs.....1729
Le Riche.....1741
Bolten.....1746
Jaegerschmid.....1765
Gooch.....1772
Greding.....1773
Baudelocque.....1777
Laflize.....1777
Murray.....1780
Cleghorn.....1784
Schuetzenkranz.....1785
Young.....1787
De Mederer, 2 cases....1787
Baillie.....1789
Dumas.....1792
Baillie.....1794
Bicker.....1794
Plouquet.....1798
Brown, 2 cases.....1798
Nysten.....1803
Merriman.....1812
Phillips.....1817
Lobstein.....1820
Hesselbach.....1820
Cruveilhier, 6 cases....1829–1835

This makes a total of 48 cases in Division 2. If to this number the cases reported by Corvinus, Cocchi, Meckel, Alquie and others are added, and also cases where only shreds were found, as by Cochon, Dupuis, Littre, and Boehmer, and in addition to these corpora lutea misinterpreted as pregnancy, and the case of Everard Home, a grand total of about 60 cases might be counted in this division, or nearly 80 cases in Group I.

In perusing the literature of ovarian tumors which contain teeth, bone or hair, the latter being at the time looked upon as products of conception, one is amused by the glib and ever ready explanation offered by those who were presumed to know. Thus, in the case of a married woman, it was due to witchcraft etc., but if found in a single woman or widow the case was one of abortive gestation, normal development having been arrested by fear, grief, or shame—consequent upon illicit intercourse. This might be termed the serio-comic side of the situation. Many a woman's reputation was blasted and her good name sacrificed on the altar of ignorance. This is not only sad to contemplate—it is tragic (see Astruc, Dezeimeris, Cauwenberghe, De Smet in Literature appended).

GROUP II.

In this group the decisive point is absence of the ovary on the affected side. In its stead a gestation sac appears, supposedly proving ovarian pregnancy. It comprises 20 cases.

Bianchi	1728	Ballard	1806
Giffard	1730	LaChapelle	1811
Boehmer	1749	LaChapelle	1816
Meckel	1758	Susewind	1820
Bland	1786	Barkhausen	1824
Krohn	1790	Guerard	1828
Petit	1798	Rahts	1828
Forrester	1798	Flachs	1837
Walter, J. G	1805	Maier	1839
Heim	1805	Dartigues	1842

GROUP III.

In this group are the cases of ovarian pregnancy, claimed as such because the organ is enlarged, frequently ruptured, but in all of them true ovarian substance is demonstrable. They number seven.

Mercer	1614	Granville	1819
Saint Maurice	1682	Hoepfner	1819
Montagnier	1697	Sammhammer	1823
Charnaux	1771		

GROUP IV.

In this group ovarian pregnancy is claimed on the ground that the ovum or some part of it is adherent to the ovary. Their number is six.

Gmelin	1781	Doudement	1824
Daynac	1823	Stanley	1810
Soemmering	1794	Velpeau	1824

GROUP V.

The cases in this group claim to have either the fetal cord or the placenta within, or upon, the ovary. They are six in number.

Heim	1793	Dubois	1831
Ucelli	1809	Menolti	1838
Novara	1814	Cruveilhier	1842

GROUP VI.

These cases make the claim that the impregnated ovum is situated within the substance of the ovary itself. They are five in number.

Littre	1701	Smith	1809
Varocquier	1756	Hesselbach	1824
Jaegerschmid	1765		

GROUP VII.

This group contains all cases which, owing to incompleteness of their description, or wrong reference, or inability to gain access to the original articles or reports, could not consistently have been included in the previous groups. For one of these reasons, therefore, they are placed here in a group by themselves in order that the record of all supposed ovarian pregnancies reported in the Literature, up to the year 1845, may be made complete.

Author	Case of	Cited on page	Year
Bolten	Mueller and Friederici	96	1747
	(*Hamburg Magaz.*, Vol. I, St. 1, 1748)		
Boehmer	Gemnil	43	1752
	(*Observ. anat, rar. fasc.* I, *et* II, *Halae Magd.*, 1752)		
Baillie		75	1789
	(*Philos. Trans.*, 1789)		
Weinknecht	Barfenknecht—Thom	2 and 3	1781
	(*De concept. extrauterina Halae*, 1791)		
Deutsch	Walter, J. G.—Blondin	57	1679
	(*Zodiacus Med. Gall. T. I., pro* 1679)		
Plouquet	Behr—Ettmuller		1794
Voigtel	Blasius	550	1805
Heim	3 cases	19–21	1812
	Wolfe and Brucker	22–24	
Home	Cruikshank	63	1819
Lobstein		137	1820
Kolpin		238	1820
Kilian	Sabatier—Moore	217	1834
Cruveilhier	Albosius		1582
	Bourdois et Chomereau		1747
	Muehlbeck		1786
	Denman		1786
Dezeimeris	August Petsch	237	1837
Ecker	7 cases	29	1841
Mayer	Camper		1759
	Kleefeld and 7 additional cases		1794
Cauwenberghe	4 cases	227	1867
De Smet	8 cases		1868
Hess	Nagel	35	1869
Schroeder	3 cases	218	1872
Schweninger	Stein	608	1875

PART II.

We have now arrived at a time in the history of ovarian pregnancy which is full of promise. Mayer has just published his epoch making essay in which he denies the possibility of ovarian pregnancy and shows that the cases reported as such are open to grave doubt. The effect of this is, that authors are more

careful and precise in the description of their cases. Let us briefly analyze the cases of ovarian pregnancy reported between the years 1845 and 1880.

Hein (1848) described the case of a woman, aged 67, who died of tuberculosis. At autopsy the right ovary was seen to contain a hard tumor the size of a hazel nut. Within this growth were the long bones and ribs of a lithopedion. These bones had not escaped from the ovary, but were enclosed by the fibrous covering of the latter. The uterus, left ovary and both tubes were practically normal. Through some inadvertence these were lost, but the fetus was preserved.

As a matter of fact, the author saw neither the uterus nor the adnexa in this case. Only the fetus was sent to him for study. It is questionable whether the one who performed the autopsy and who neglected to preserve the uterus and adnexa was capable of forming an authoritative opinion as to the presence of ovarian pregnancy. We merely have his statement that the "adnexa were practically normal." The most one can say for this case is that it has not been proved.

Kiwisch (1852) does not believe in the cases of ovarian pregnancy recorded up to his time. However, he will not go so far as Mayer, who denies the possibility of their occurrence. To prove how easily one may be misled into describing cases as ovarian pregnancy if one judges only by absence of the ovary on that side, he cites a specimen so labeled in the pathological Institute in Würzburg, in 1849. Professor Kölliker dissected the specimen and, being an expert anatomist, finally succeeded in bringing to light a very much atrophied ovary, which had been buried among a dense network of adhesions.

The case of ovarian pregnancy which this author described may, without hesitation, be characterized as not proven.

Virchow says that he himself is not sure that the case he reported is one of true ovarian pregnancy. He says it is probably so.

Reymert (1852).—The left ovary, the size of a goose egg, was surrounded by blood clot. The abdominal cavity was filled with clotted blood. On removing the latter, an embryo three inches long was found in the abdominal cavity. The membranes surrounded the fetus and its cord was attached to the ovary.

From these meagre details it would be impossible to form a just opinion of the case. As the burden of proof lies on the author, one is forced to admit that, as far as the evidence went, the claim to the title of ovarian pregnancy has not been established.

Sinclair (1853).—A woman, aged 36, and the mother of four children, died in the fifth month of her fifth pregnancy. A small quantity of blood was found in the pelvis, which was occupied by a considerable tumor. Near the left sacro-iliac synchondrosis was a small rent beneath the clot, and this led into a cyst, which contained a fetus of considerable size. The uterus was displaced and formed the roof of the cyst. On the left side the corresponding tube, round ligament and ovary were found. The latter was transformed into a yellowish-brown, pultaceous mass. On the right side a portion of the Fallopian tube and round ligament was seen, but no trace of an ovary. In its stead was a large fibrous cyst. The fetus was connected by its cord to the placenta which, in turn, was attached to the fibrous cyst already mentioned and which was situated on the right side.

This case is obviously not one of ovarian pregnancy. It would, therefore, be a waste of time and space to bring proofs. A number of authorities agree on this point.

Uhde (1857).—The patient was the mother of two children, and died 11 weeks after cessation of the last menstruation. At autopsy the abdominal organs were seen to be generally adherent to each other. At the bottom of a cavity formed by adhesions and which contained some fluid blood, the right ovary was found. The latter was enlarged and emphysematous, and at its posterior surface and behind, it was connected with a sac which was formed of chorion. This sac approached in size that of a large plum and had a rent on its posterior surface. The sac contained an embryo, about seven to eight lines in length, surrounded by villi, and showed an umbilical vesicle. Right tube and fimbriæ free. Uterus four inches long and three inches broad. Its cavity contained a decidua.

According to Gurgui, this is a case of secondary implantation upon the ovary. Some essential details are lacking. In the first place it is rather difficult to understand how there could be a general adherence and matting together of all the abdominal organs (no doubt due to a general peritonitis) and only just this right tube and even its fimbriæ "free." This is the more extraordinary when it is considered that the ovary had to be dug out of a bed of adhesions, at the bottom of which it was found, and nevertheless its tube and even the fimbriæ were "free." Another interesting fact is that, although the sac was torn, the embryo still remained within. Finally, an embryo of eleven weeks ought to be more than 7 to 8 lines long. No good purpose can be served by continuing the critique of this case, but it may safely be assumed that it is not one of ovarian pregnancy.

Helie (1857).—The author himself cites this case as one of extra-uterine pregnancy. Later writers persistently quote it as ovarian. It is mentioned merely to be put under this caption and dismissed.

Hecker (1858).—This patient was 28 years old. She had borne one child. Three years later, while in the 18th or 20th week of her second pregnancy, she suddenly expired. Autopsy.

The author will not say positively that this is an ovarian pregnancy, but that it awoke in him a subjective conviction that the case was of such nature. It would seem that, in view of the fact that neither the tube nor the placenta was ever found or accounted for, the only subjective conviction the case should have awakened in him is, that it was not one of ovarian pregnancy.

Willigk (1859).—The case of ovarian pregnancy reported here seem to be a favorite with authors and is almost universally accepted as such. It is no less than the report of specimen No. 179, of the Pathological Museum at Olmütz. The salient points for us are: The right adnexa are normal. The uterus slightly enlarged. The two lamellæ of the left broad ligament are spread apart and enclose a sac, 7 cm. long, which has a small tear in it. The sac contains an embryo 4.5 c.m. long and estimated to be of about three months' gestation. The sac ends at one point in the ovary, which has become enlarged to 5 cm. in length.

It requires no lengthy argument to point out that a fetus found within the layers of the broad ligament is intraligamentary. It must follow that the pregnancy was intraligamentary, having, as the author puts it, " spread apart the two lamellæ of the left broad ligament." It does not concern us that the pregnancy may have been at one time ovarian. We are dealing merely with the conditions as they were found by the author who reported the case. From his own statement, then, this is surely not a case of ovarian pregnancy.

Walter (1861).—The patient was a married woman, 40 years old. She had borne three children. The last coitus was on August 31, 1857. She was then menstruating at her regular time. The next day she left on a journey of 26 miles by wagon. The conveyance was accidentally overturned, and she was thrown into the snow (*sic*). She had to assist in righting the wagon and exerted herself considerably.

Menstruation did not occur again. She suffered repeatedly from abdominal cramps. The following May the author saw her and made out a rounded mass in the pelvis. In August a fetus could be distinctly outlined in the abdomen. Two months before that time she felt fetal movements. A diagnosis of extra-uterine pregnancy was made. On the 12th of October peritonitis developed, which caused her death two weeks later. The autopsy showed signs of general peritonitis. The child was well developed and weighed six lbs. The placenta appeared to be enclosed in the right ovary, which was greatly enlarged. The right ovarian ligament was twice its normal size. The uterus and left adnexa were practically normal. The right tube and fimbriated extremity were quite normal and non-adherent. The oval mass, which was taken for the right ovary, contained a large amount of placental tissue. However, in no part of the supposed ovary was ovarian tissue demonstrable.

A proper critique of this case, which is remarkable in many ways, would take up too much space. Therefore, the question of the discrepancy of dates cannot be gone into, for instance: If on the 31st day of August, 1857, fruitful intercourse was had, a rounded mass would be felt in the pelvis long before the following May—almost nine months later. Likewise, the first fetal movements were felt one month later, namely, about 10 months after the supposedly successful coitus. In August, 1858, or one year later, the fetus could be distinctly outlined and the diagnosis of extrauterine pregnancy made. The one important point in this case is the fact that, although an expert examined the specimen, he had to admit that in no part of the supposed ovary could even a faint trace of ovarian substance be demonstrated. It is unfortunate that a snow storm of such severity as to have been the cause of upsetting the wagon in which the lady rode, should have occurred in August. While it might be possible to convince one that heavy snow storms occur in August, it is asking too much of anyone to accept this case as an ovarian pregnancy.

Wiedersperg (1865).—Patient 34 years old. Last menses four months before death.

The most prominent fact appears to be that the Fallopian tube on the affected side ended blindly in and was lost within the gestation sac. The case is, therefore, not one of ovarian pregnancy, but probably tubal or tubo-ovarian.

Horwitz (1868).—Woman, 35 years old, who had four children, died in the fourth month of her fifth pregnancy.

Schroeder stoutly maintains that this is a true ovarian pregnancy. In the light of our present knowledge, however, especially as details of this case are wofully lacking, particularly as regards the tube on the corresponding side, we cannot accept this as a case of true ovarian pregnancy.

WRIGHT (1869).—Woman of 36, mother of two children, died in the fourth month of gestation.

The author does not claim this as a case of ovarian pregnancy, being content to name it extrauterine. Schroeder, in this case also, insists that it is ovarian; hence, it has been mentioned here. It may be said that the author, who saw the case, ought to know, and, probably, is right.

HESS (1869).—Woman, aged 37, 2-para, died in the eighth month of her third pregnancy. At autopsy a tumor the size of a man's head was found. Also signs of general purulent peritonitis. The left ovary was absent and the outer third of the left tube was lost in the gestation sac.

This is, therefore, a case of tubo-ovarian, not ovarian, pregnancy.

ORTH (1874).—At a meeting of the *Gesellschaft f. Geburtsh. und Gynec.* in Berlin, held in May, 1874, the author presented two specimens obtained at autopsy. Both were cases of extra-uterine pregnancy—one of three months' and the other of four months' gestation. Case 1. The left ovary, about twice the size of a man's fist, contains an embryo 16 cm. long and a cord 32 cm. in length. Death of the patient was caused by rupture and internal hemorrhage. Case 2. Condition of chronic adhesive peritonitis which was so intense that it caused a conglomeration of all the pelvic organs into an inextricable mass. It was quite impossible to get the ovaries and tubes free. The gestation-sac was situated in the right iliac fossa.

These cases, often quoted as ovarian pregnancy, have in reality very little claim to that appellation. Under the circumstances a critical review is superfluous.

SCHWENINGER (1875).—Patient aged 35 or 38 years. After 12 years of married life she became pregnant. The pregnancy lasted three and a half years and ended fatally. At autopsy signs of general purulent peritonitis were found, also universal adhesions between the abdominal organs. In the right lower quadrant of the abdomen was a large adherent sac, which was torn in three places and contained a quantity of foul-smelling pus and the small bones of a full-term macerated fetus. The right ovary could not be found. The right tube runs along the

upper border of the sac, and its fimbriated end, while in the wall of the sac, does not communicate with the interior of the sac. The right round ligament is easily discernible, The left ovary, round ligament and tube are fairly normal.

This case, although looked upon by some authors as an ovarian pregnancy, cannot sustain its claim to that title if viewed in the light of our present knowledge. In the first place, here is a sac of large size, containing a quantity of foul-smelling pus and some small bones of a fetus. There is no evidence brought forward that this is really an ovarian sac, except the bald statement that the ovary on that side was not seen. It is not unlikely that the ovary was there, but so covered by the adhesions of a general peritonitis that it escaped notice. Such cases are on record. There was no microscopical proof adduced that ovarian tissue, or something even remotely suggesting ovarian stroma, was seen in any part of the sac. It might be objected that after a pregnancy had lasted three and one-half years and the products of conception had become purulent, ovarian structure could hardly be expected to be demonstrable. Allowing this objection to stand for the sake of argument, one goes a step further and is confronted by the fact, that the sac is not connected to the uterus by the ovarian ligament. This is one of the cardinal signs and cannot be ignored. Upon this alone, absence of ovarian pregnancy may be positively asserted. There is one more point which ought to receive some attention. The Fallopian tube on the affected side runs along the sac wall and its fimbriated end is lost in the sac. That fact of itself is, according to the best authorities, sufficient to annul any claim to the title of ovarian pregnancy; it also proves the charge made that if authors would take the trouble to investigate each case instead of (as sometimes happens) merely copying the opinions of previous writers, mistakes of this nature would not be perpetuated.

Landau. Leopold (1879).—A woman aged 36 years. Twelve years before she bore a child. Since then she had a miscarriage at three months. Twelve months after the last menstrual period she had severe abdominal pains and gave evidence of peritonitis. The author diagnosticated abdominal pregnancy. At the laparotomy a fully developed dead child was extracted. The placenta was not removed, but sewn into the wound. Except that she had slight fever, some suppuration and several attacks of secondary hemorrhage from the site of the placenta, the patient did fairly well for five weeks after the operation. She finally died of pulmonary edema.

The very complete report of the autopsy shows that the Fallopian tube on the left side became lost in the sac wall. The ovarian ligament was *not found*, therefore, could not be said to connect the sac to the uterus. Furthermore, the left ovary was distinctly seen within the gestation sac. It is, therefore, difficult to understand how, in the face of such a report, the author and others following him, could interpret this as a case of ovarian pregnancy.

Puech (1879).—A woman, who had been murdered, came to autopsy. In her left ovary a body of round shape and 2 cm. in length was noticed. With aid of a magnifying glass, an embryonal deposit about 1 mm. in size could be made out.

The description of this case leaves the reader unconvinced, because the necessary microscopical proof was not furnished.

Collet y Gurgui (1880).—This is the report of a case of ovarian pregnancy. The author did not see the patient, but received the specimen from another physician who had performed the autopsy. The specimen consisted of a ruptured gestation sac which contained a fetus, the left ovary, epoöphoron, Fallopian tube with its fimbriæ, and the remains of the broad ligament. This specimen was removed from a woman 33 years old, the mother of two children. She had died suddenly of an internal hemorrhage. At autopsy a large quantity of fluid blood was found in the abdominal cavity, and what was taken to be a left-sided tubal pregnancy removed. The uterus and adnexa were not removed because the husband objected.

If there were any desire to be hypercritical, astonishment might be expressed that a husband, who evidently had consented to an autopsy on his wife, and permitted the removal of her left ovary and tube, together with its fimbriæ, the epoophoron, and also what remained of the broad ligament, should positively refuse to have her uterus and right adnexa removed. Accepting this as a fact, we find, on reading the somewhat voluminous report, that there was no connection of the sac to the uterus by the ovarian ligament, the tube was intimately adherent to the sac, and that no ovarian tissue was demonstrated in the sac wall. The case can, therefore, not be accepted as one of ovarian pregnancy. Finally, the author himself admits that this case can just as well be interpreted as one of abdominal pregnancy.

This finishes the study of all cases of alleged ovarian pregnancy in the medical literature up to year 1880, as far as they have been accessible.

We may now glance at the cases reported in the medical literature as true ovarian pregnancy from 1880 to 1903. At this point I desire to acknowledge my indebtedness to Kantorowitz, who, in a very able article in Volkman's Sammlung Klinischer Vorträge, in 1903, gathered the more important cases of this nature up to his day and added two cases of his own. In working up the material during this time, I shall follow Kantorowitz closely. The first is one reported by

Leopold (1882).—This woman was 75 years old and had been pregnant eight times. Shortly after the birth of her fourth child and when she was 40 years of age, she conceived again. This time she gave signs of extrauterine pregnancy. About the normal end of gestation the fetus died and gave no further trouble. The three children which were born later were all delivered spontaneously and the puerperia were normal. In the right lower abdominal quadrant she had a tumor which was diagnosticated as a lithopedion. It grew harder and more solid each year, but this did not prevent her from reaching a ripe old age. There is no record of the autopsy. Here is a brief description of the specimen: The tumor weighed 1550 gm.; length, 17 cm.; breadth, 15.5 cm.; greatest circumference, 41.2 cm.; smallest circumference, 31 cm. The fetus was a female and was about 44 cm. long. The tumor showed the following connections with neighboring organs: Above and to the left, to the great omentum; below and to the left, the wholly free right tube and the right lig. ovarii. The tube runs along the tumor for 3 or 4 cm. The ovary on that side is absent. There was no microscopical examination of the sac wall.

This most rare and interesting case deserves more than passing notice. It is really difficult to decide the exact nature of this pregnancy. In the first place, the tube is not described with the accuracy and the detail expected in such a report. Nothing is said specifically of the fimbriated extremity; yet this very point is of vital importance in differentiating between a tubo-ovarian and purely ovarian pregnancy. Secondly, no microscopical proof has been presented to show the ovarian origin of the sac or some part of it. Thirdly, although all the organs of the fetus, even its umbilical cord, are said to be beautifully preserved, the placenta cannot be found at all. It is *assumed* by the author that a thickened portion of the sac wall is the seat of the whilom placenta. Furthermore, we read that the omentum was extensively adherent to the fetus and fed the latter by means of large blood-vessels. These prevented its disintegration. While, on the one hand, it would be most

difficult to prove the original implantation of the ovum after so many years have elapsed, on the other hand, it must be admitted that this has not been proved to be a case of true ovarian pregnancy and, therefore, cannot be accepted as such.

STRATZ (1890).—This woman was a II-para and had gone through the two labors without special assistance. About a year after the last confinement she gave the usual signs of gravidity complicated with symptoms of extrauterine pregnancy. She went along in this way for about four months, when she consulted the author about the middle of May, 1889. Upon examination the uterus was found to be soft, enlarged and anteflexed. Behind the uterus a soft diffuse swelling ran along the left broad ligament. At operation the following condition was found: Uterus soft, movable, relaxed. Right adnexa normal. Left tube thickened. Under this tube a red cauliflower-like mass is visible. This appears to be on top of the left ovary and protrudes into the abdomen. It looks like a blood coagulum. This diffuse growth which extends into the cul-de-sac of Douglas is allowed to remain, but the uterus and adnexa are removed. The patient recovered. Two months later the mass had disappeared.

Hardened in alcohol, the tube is found not to be greatly enlarged. Tube and ovary free. No fetus can be found. Microscopically the tube is normal. The ovary shows myxomatous degeneration in places, also chorionic villi which are the site of myxomatous degeneration.

As this case is almost universally quoted as one of undoubted ovarian pregnancy, it has been gone into rather more fully than most of those preceding it. Reasons for having some doubts about it are: 1. The tube was not normal, even though it may appear to have been so at the microscopical examination. It had been hardened in alcohol and yet was by this process shrunk down to a size which the author calls *not greatly enlarged.* 2. Not even one normal chorionic villus was demonstrable in the ovary. 3. The ovarian ligament was not shown to be directly connected with the sac. 4. No fetus or the point of its implantation was to be found anywhere. 5. There was a large mass (placenta ?) which was not examined or removed at the operation and which remains unaccounted for. When it is recalled that this mass was located immediately below and in juxtaposition to the tube in question, one's suspicions become strengthened that this can not well have been a true ovarian pregnancy.

ROSTHORN (1890).—This is a case cited by very many authorities as an example of true ovarian pregnancy.

That this is not the fact requires only a glance at page 419 where, in summarizing*, the author says: " In this case ovarian pregnancy is excluded from the beginning, because of the implication of the distal end of the tube on that side in the formation of the gestation sac."

Saenger (1890) reported a case of " ovarico-abdominal pregnancy." The woman was 39 years of age, and the author operated on her for a suspected abdominal pregnancy. Microscopical examination failed to reveal ovarian tissue in any part of the sac.

Without further description, it is plain that this was not an ovarian pregnancy. It is included here principally because it has been so frequently cited as an authentic case.

Wyder (1891).—Woman, 28 years of age, died from septic peritonitis following laparotomy for extrauterine pregnancy. Upon microscopical investigation neither chorionic villi nor ovarian stroma could be found in the wall of the sac.

In this case, as in the one preceding it, ovarian pregnancy obviously does not exist; it is inserted here for a similar reason.

Herzfeld (1891).—A 33-year-old peasant woman had borne two children and experienced one miscarriage. This time she was delivered at term, and the author, having recognized an abdominal pregnancy, performed laparotomy. Immediately upon opening the abdomen a tumor which resembled an ovarian cyst presented. This turned out to be the right ovary. It contained a quantity of meconium, a little liquor amnii, and a male fetus which had died at an early period.

As no ovarian tissue was found in the gestation sac, this case cannot be accepted as one of ovarian pregnancy.

Mackenrodt (1891).—At a meeting of the Berlin Obstetrical and Gynecological Society, held in November, 1891, the author reported a case of ovarian pregnancy as follows: Woman, 32 years old, was always regular. Since a seven-weeks' amenorrhea she has been bleeding irregularly. A soft tumor, the size of a goose egg, was made out under anesthesia. Curettage produced masses of soft endometrium which did not show the characteristics of decidua. The diagnosis made was extrauterine pregnancy. Ten days later the tumor increased rapidly in size, gave pain, and caused some rise in temperature. At laparotomy the tumor was found to be very adherent, but had a small pedicle. It burst during removal and discharged a considerable amount of fluid blood, also old coagula. The fetus could not be found. The tube appeared fairly normal in size for about 2.5 cm., then it rapidly grew larger until it was the size of two thumbs. Just before reaching its fimbriated ex-

* See appended Literature.

tremity it contracted in size to normal. The fimbriated extremity is lost in a cystic ovarian tumor the size of a hen's egg. The latter burst during removal. The ovarian tumor can be well isolated from the tube and in its lower portion, which contains ovarian tissue, a bean-sized corpus luteum is visible.

Time and space forbid a full discussion of this most interesting case. Attention will be directed to only one of the cardinal points which must necessarily be established before a case may be characterized as one of ovarian pregnancy, namely, the tube on the corresponding side must be free from the sac and normal in every way. This was not the case here. This case cannot be designated as one of true ovarian pregnancy.

Kouwer (1893).—Woman, 31 years of age, V-para, missed her period six weeks and gave signs of internal hemorrhage. Diagnosis ectopic pregnancy. Laparotomy was done forthwith. The right adnexa were removed, and appeared as follows: The tube was perfectly normal and free and isolated from the ovary by a long mesovarium. The lumen of the tube was open and showed no pathological adhesions. The ovary was considerably enlarged. Broadly connected with the ovary was a walnut-sized, brownish red tumor, the outer covering of which was continuous with the covering of the ovary. On the pole of the tumor was seen a rough surface, as if adhesions had been broken here. In a little excrescence adjoining the pole of the tumor was a small opening which, during the operation, was found filled by a blood coagulum, and from which, after the specimen had been preserved in strong alcohol, reddish fringes protruded. A median section through the tumor and which passes through this opening bisects the gestation-sac lying within the tumor and also divides the partly macerated, brownish discolored fetus, which is about 12 mm. long, upon which the cephalic and caudal ends as well as the rudimentary extremities are visible. At the place where the gestation-sac borders upon the ovary, a loosely-woven tissue is seen which impresses one as being placenta. Microscopically chorionic villi are seen developed in the whole periphery of the gestation-sac, and the intervillous spaces contain blood coagula. The perforation did not lead into the gestation-sac, but into the blood sinuses. The fringes which protruded were villi. The opening was surrounded by a thick layer of connective tissue. The covering of the sac towards the ovary consists of a layer of connective tissue cells which, in spite of their greater or lesser change into necrotic decidual cells, showed the arrangement typical for the Theca Folliculi. The ovary itself shows Graafian follicles in various stages of development, the only abnormality being advanced hyalin degeneration of the vessel walls and a few small cysts.
For discussion of this report see Van Tussenbroek. (See p. 262).

Martin (1893).—At the fifth Congress of the German Gynecological Society, held in Breslau, in 1893, the author demonstrated a specimen of ovarian pregnancy. Twenty years before operation, the diagnosis of

extrauterine pregnancy had been made. At that time the fetus died and the patient suffered a severe attack of peritonitis. Seventeen years later she got carcinoma of the cervix uteri, and it is for this condition that laparotomy was performed. At operation the gestation-sac was removed; also the uterus and the adnexa. The patient recovered. The fetus, which was converted into a lithopedion, was situated in the left ovary. The specimen shows the uterus, right ovary and both tubes.

The demonstration does not show the left tube to be quite free from the gestation-sac. No microscopical proof was submitted to support the claim made. The case cannot, therefore, be accepted as one of true ovarian pregnancy. The other specimen shown by the same author at the same meeting, is so obviously a tubo-ovarian pregnancy that its description would be out of place in this paper.

Gottschalk (1893).—At a meeting of the Obstetrical and Gynecological Society, in 1893, this author presented a well-preserved lithopedion weighing about five pounds. He had obtained the specimen by laparotomy from a woman 54 years of age, who had carried it for 30 years. He said: "The whole gestation-sac is surrounded by a closed ovarian capsule which varies in thickness from one to several millimeters. In this outer capsule or covering, numerous corpora lutea and Graafian follicles are visible. Under this outer covering of ovarian tissue is one of greater thickness and chalky composition. Within this chalky capsule was the five-pound fetus, and together they made a tumor which reached up to the woman's umbilicus. The corresponding tube was elongated but otherwise normal."

The temptation to discuss this case in its various phases is almost irresistible; that would lead us too far, however. It may be pointed out that, in all probability, the case was not one of ovarian pregnancy, because the sac was not shown to be connected to the uterus by the ovarian ligament.

Ludwig (1896).—In February, 1896, a woman, 35 years of age, who had borne six children, gave birth to a female child. Following this event the midwife noticed another child moving within the patient's abdomen. Six days post partum the patient undertook a 17-hour journey to Vienna. Here a laparotomy was done by the author and a living male child extracted. An elastic ligature was placed around the lower part of the uterus and the latter removed. Mother and child left the hospital four weeks later in good condition.

Professor Kolisko examined the specimen and made a very careful and exhaustive report. He gave it as his opinion that the case was one of ovarian pregnancy. Some doubt as to the correctness of this conclusion must arise in the mind of

the reader of that report for two reasons: Firstly, no microscopical investigation of the tube on that side was made; therefore, the possibility of the case having originated as a tubal abortion and becoming secondarily an abdominal pregnancy cannot be excluded. Secondly, the histological picture of the ovary was that seen in the unimpregnated condition. It looked like, and probably was, normal ovarian tissue. It did not show the histological changes met with in a pregnant ovary, namely, heaping up of cells, hemorrhages, chorionic villi. This case can not, therefore, be cited as having been proved ovarian pregnancy.

Toth (1896).—This case, described as one of ovarian pregnancy, occurred in a woman 32 years of age, who had borne one child at term previously. She carried the fetus to term.

As this report, otherwise very full and complete, fails to show that the gestation-sac was attached to the uterus by the ovarian ligament, one of the most important proofs of ovarian pregnancy is missing. For that reason, this case cannot be classed as one of ovarian pregnancy.

Tussenbroek (1899).—At a meeting of the Gynecological Society of the Netherlands, in April, 1893, Kouwer, of Haarlem, reported a case of ovarian pregnancy which he had operated on after rupture. For several years I was compelled by the necessities of my daily practice to neglect the examination of this extraordinary specimen. This article to-day is an effort to acquit myself of the old debt and as an appreciation of the honor done me by entrusting me with this investigation. This is not, therefore, a premature publication. The tissue was cut with a rocking microtome, and that may account for the difficulties in the technique. With this instrument it is almost impossible to cut sections in a flat plane. After a lot of trouble three small series of sections were obtained. . . . The specimen was hardened in Mueller's fluid and later in alcohol. It consists of the right tube and ovary. The tube is perfectly normal. The fimbriæ are somewhat glued together, but the lumen is open. No pathological adhesions between the tube and ovary. Upon the ovary a small, round prominence is noticeable. Near the upper pole of this a perforation is seen, from which reddish fimbriæ protrude. A cut was made through the ovary and tumor in such a way that it went through the site of perforation. The section disclosed the fact that the tumor was entirely composed of a cavity which was attached by a broad base to the ovary. This cavity contained an embryo, about 12 mm. long, which was attached to the gestation-sac by a short, thick cord. The microscopical examination shows that the product of gestation is situated within a Graafian follicle. At the junction of the gestation-sac and the ovary, a formation of tissue is seen in the microscopical

picture which shows perfectly the edge of a corpus luteum. One can recognize in it the peculiarly convoluted wall of lutein cells, and outside of this the theca externa with its vascular wreath. From the theca externa loops of vessels crowd forward between the folds of lutein tissue. Upon examination of other parts of the gestation-sac, remains of the lutein wall are plainly seen everywhere. These remains are positively recognizable as such, close up the site of rupture; in places where the gestation-sac was thinned to a mere membrane, they were crushed and distorted. This proves that the ovulum lay in a Graafian follicle which, after bursting, became converted into a corpus luteum.

As Kouwer's report describes the same case, it will perhaps be best, in order that time and space be conserved, to consider them here together. Both authors are to be congratulated—Professor Kouwer for correct diagnosis and successful operation, and Dr. Catharine Van Tussenbroek upon her exceedingly able and elegantly written article. The case in question was received by a great many of the best authorities on the subject as one of proved ovarian pregnancy and to this day is cited as such by writers of text books. Unfortunately, it oftimes happens that in so doing the opinions of previous authors are merely copied into the new book, thus perpetuating even errors in references to the literature. It is needless to state that such practices, while they are doubtless timesaving, are not exactly conducive to real advance in the science. If, therefore, one questions the conclusions arrived at by a previous investigator and does a little thinking of his own, in an humble spirit and merely as a student seeking after truth, it is hoped that this course will not be criticized as presumption. In the first place, Professor Kouwer states: "At the place where the gestation-sac borders upon the ovary, a loosely-woven tissue is seen which impresses one as placenta." It is superfluous to say that a gynecologist of world-wide reputation knows placental tissue when he sees it. Therefore, we need not hesitate to accept his assertion that the tissue situated on the outside of the mass where the tumor adjoined the ovary was, as it appeared to him to be, placenta. This is a vital point in determining the implantation of the ovum, and unless explained away would tend to prove that the case was one of secondary implantation on top of the ovary, after having been imbedded primarily somewhere else. The pathologist who made the microscopical examination of the specimen does not enlighten us on that point and does not even mention

the subject. Dr. Van Tussenbroek did not publish the result of this investigation until nearly six years after the operation, so that it could not well be said to have been overlooked in the hurry of a superficial examination. Neither can it be assumed that such a careful and painstaking investigator who, in a report which is a model of accuracy as to detail, in which the amount of protoplasm of a cell and the shape of a cell nucleus, also the degree to which a cell protoplasm takes up the color of a particular stain is given vivid description, should have been guilty of an oversight which would be fatal to the description of her specimen. One is, therefore, quite at a loss to understand why this particular piece of tissue should have escaped the microtome when the doctor was cutting the ovary into serial sections. Both authors assert that the tube on the affected side was normal. On further investigation one finds that the fimbriated extremity of this Fallopian tube was not exactly normal but that, as they express it, the fimbriæ were *somewhat glued or matted together*. Ordinarily this would not mean much in the way of pathology. However, the significance of finding such an abnormal condition of the tube is vastly greater in a case of this nature, because it is upon this very point, namely, a perfectly normal condition of the tube, that the whole question of ovarian pregnancy hinges. Perhaps this may have been the reason which induced Professor Leopold Meyer, of Copenhagen, to object that this may have been a tubo-ovarian pregnancy rather than a purely ovarian one, when the doctor exhibited the specimen and read the report of this case. Furthermore, in his original report of this case, Professor Kouwer stated that, on the pole of the tumor, a rough surface was visible, *as if adhesions had been torn loose there*. In her paper the doctor does not describe this condition at all; in fact, does not mention it in any way. It does not take a very great amount of discernment to see the connection of this unexplained raw surface, which looked as if adhesions had been torn loose there, and the matted, pathologically changed fimbriated extremity of the Fallopian tube on that side. It does not seem impossible for these to have been connected and torn apart during the manipulation necessary during operation. Nothing seems more natural in fact, than the loosening of adhesions by the fingers of the hand introduced within the abdomen, while groping in the dark of a pelvis filled

with blood, in the endeavor rapidly to remove the mass in a ruptured ectopic. Finally, there is in this article one thing more than any other, which is calculated to raise doubts in the reader's mind as to the character of this pregnancy, and that is, the beautifully clear and classical description of the Graafian follicle in which this ovum is supposed to have been imbedded and developed for about six weeks. The most minute details of cells of the Graafian follicle and corpus luteum are described with a realism and precision that might be the envy and despair of a text book on normal histology. It is no wonder that some parts showed degenerative changes when it is considered that the specimen had lain in a jar for a number of years before it was cut for microscopical examination. The doubt which forces itself upon the reader almost against his will is this: is it possible for cells and tissues to maintain their identity when in close proximity to a growing ovum? Is it not a fact that when the ovum becomes imbedded in a tissue and the trophoblast begins to eat its way into the maternal parts, it opens up maternal vessels and causes blood to be effused into which the villi dip for nutriment? That this process goes on for a considerable distance around the nidus, and that this effusion of blood makes the finer histological characters of tissues and cells unrecognizable? If this is true (and that it is, we need but refer to text-books on the subject), it must follow that it would have been manifestly impossible to distinguish lutein cells or, for that matter, any other kind of cell, when the whole gestation-sac was perforated by villi of the ovum contained within. One is thereby brought reluctantly to one of two conclusions: 1. Either the author was mistaken and the Graafian follicle which contained the ovum did not show all the described characteristics of a normal or nearly normal condition, or 2. The condition was exactly as described and that proves that the follicle in question was not the seat of the primary implantation of the ovum. It goes without saying that the latter of these two propositions is the one to be accepted. There are some other points that would require further explanation, such for instance, as the more exact description of the umbilical cord and its site of attachment, etc. Sufficient has been said, however, to indicate that, while the possibility of this case having been one of ovarian pregnancy is not absolutely denied, yet, if accepted as such, respect for the

opinion and position of the distinguished authors would have more weight in this direction than the evidence brought forward in support of this claim.

Füth (1902).—This was a woman, 25 years old, who alleged that she was pregnant for the first time. Later, however, she admitted that there was a possibility of her having been impregnated three years before. Laparotomy disclosed a lithopedion in the left kidney region. This fetus corresponded to one of about five months. The uterus and left adnexa were normal. The right tube appeared to be normal, but was not examined microscopically. A fist-sized tumor was seen in the location of the right ovary. The above-mentioned tumor consisted of a main mass connected by a thick pedicle to the smaller mass, which was later shown to be the right ovary. The petrified fetus was everywhere adherent to the great omentum. Its cord could not be found. The larger of the two masses was apparently a partly calcified placenta which had become attached to the right ovary. The latter was so well preserved that a well defined fresh corpus luteum was visible on its surface near the fimbriated extremity of the Fallopian tube.

In this case of lithopedion the original place of imbedding of the ovum must necessarily remain a matter of speculation. In the first place, it was found in the *left* kidney region extensively adherent to the great omentum. It is supposed to have been at first attached to the *right* ovary. It, therefore, had to travel some distance before becoming attached in its new quarters, at the furthest possible point from its original location. Secondly, there is no evidence brought forward that the ovary had any part in the formation of the gestation-sac. On the contrary, it is stated that the mass which was looked upon as a partly calcified placenta, was attached to the well-preserved ovary by a pedicle. At best, therefore, if this case must be interpreted as one of ovarian pregnancy, it might, perhaps, have been one of secondary implantation on top of the ovary, and in that case not a true ovarian pregnancy at all. That the Fallopian tube on that side was not examined microscopically, is very unfortunate, because by reason of this omission one cannot positively deny the existence of signs of pregnancy in that tube. With such very important data missing, we can neither affirm nor deny the presence of ovarian pregnancy in a given case. This seems to be precisely the situation here.

Micholitsch (1903).—This author reports two cases: one operated on by Professor Wertheim of Vienna, and the other one by himself.

Case I.—A I-para 32 years old was operated on per vaginam. The

right tube and a tumor the size of a small apple were removed. The right ovary was not seen. The tube was decidedly convoluted and its fimbriated end showed the mucosa deeply suffused with blood. Otherwise it was perfectly normal macroscopically and microscopically. The tumor was whitish and smooth. On section it disclosed an encapsulated hematoma containing a hazel-nut sized cavity. The capsule showed ovarian structure, also chorionic villi. No embryo or any part of a fetal cord was visible.

Case II.—A II-para 23 years old had the last menstrual period September 24, 1902. About two months later she passed a "skin" and this was followed by uterine hemorrhage. A tumor the size of a man's fist could be made out in the cul-de-sac of Douglas. Operation was performed by vaginal section. The left tube and ovarian tumor were removed. The former was found to be normal. Black blood, due to an old hematocele, and shreds of tissue were also removed. The tumor tore loose from its attachments during the enucleation, but had been attached to the ovarian ligament. Definite ovarian tissue was found in the sac, also masses of villi, part of which were the subject of advanced degeneration. No mention is made of a fetus or its cord.

The first thing that strikes the reader of this report is the fact the vaginal route is not, as a rule, the best way to operate for ectopic pregnancies. In a paper of this kind one cannot well go into the pros and cons of this question, as that would lead too far. Only one point will be mentioned, namely: the technical difficulties which are apt to be encountered are of such a nature that, to say the least, the specimen will have been dragged upon so much during its removal, that its original connections will become in a great measure a matter of conjecture. Witness case two in which, even in the expert hands of Micholitsch, who is an ardent advocate of this method of operating, the ovary was torn from its ligament. As neither a fetus nor any part of its cord was found in either case, they cannot well be said to represent unassailable instances of ovarian pregnancy, notwithstanding the fact (which is admitted) that ovarian tissue and chorionic villi were found in the wall of the gestation-sac.

Kantorowicz (1903).—The author, in a very valuable contribution to the history of ovarian pregnancy, reports two cases.

The first is a IV-para 30 years of age, whose last menstruation occurred about the middle of August, 1902. She was operated on October 18, 1902. The left tube, ovary and ovarian ligament were removed. The tube was found to be almost normal, macroscopically and microscopically. Only the mucous membrane was found slightly thickened by some inflammatory deposit. The sac showed ovarian tissue, but did not contain any demonstrable fetal elements.

The second case is a I-para 25 years old. Last menses August, 1902. She gave a history of bleeding for six weeks. Laparotomy was performed November 22, 1902. On the right side in the location of the ovary a pear-shaped tumor was found. The corresponding tube, as thick as a lead pencil, can be followed for about 3 cm. It is lost in the tumor, and forms a blood cyst. The ovary contained no hemorrhages. A cavity in its centre was found to be lined with decidua cells. Distinct ovarian tissue was found, but no chorionic villi. Fetus was not found.

It is rather difficult to understand upon what grounds the author bases his diagnosis of ovarian pregnancy in these two cases. The only one the reader can recognize as such is the fact that these women missed a menstrual period. No attempt is made to assert that a fetus was seen or anything suggesting fetal elements found on microscopical investigation. It would, therefore, seem unprofitable to further discuss these two cases.

Croft (1900).—A woman, 25 years of age, who had given birth to two children, died 12 days after a laparotomy. Cause of death unknown. At the autopsy no signs of peritonitis were found.

It is positively refreshing to note the absolute fairness and candor which pervades this article. No attempt is made to exaggerate, and the modesty, characteristic of the English, is so apparent as to compel admiration. The author does not insist that his case is undoubtedly one of ovarian pregnancy. He merely suggests that it may be so. However, as the evidence on this point is by no means conclusive, one is forced not to accept it as such.

Boesebeech (1904).—A woman, 41 years of age, who had borne two children, was admitted to the hospital in June, 1903. A cystic growth the size of a man's fist was found, upon vaginal examination, to be connected with the right side of the uterus. There was an irregular menstrual history. No decidual membrane had been passed. Diagnosis, ovarian cyst on right side with twisted pedicle. At the laparotomy a parovarian cyst, without adhesions and without a twisted pedicle, was removed. The corresponding ovary was twice its normal size and had a rupture, covered by a blood coagulum, which gave vent to a viscid, sanguinolent fluid. The right ovary also was removed. The left adnexa were normal. The very minute description of the macroscopical and microscopical appearances shows that neither a fetus nor a gestation-sac was found.

In this case the author tries to draw an analogy between the appearances found here and those described by Van Tussenbroek in Kouwer's case. The cavity in which the fetus is at one time supposed to have been, is described in similar fashion. This cavity shows, among other things, a number of degenerated but

still recognizable chorionic villi, which lie here and there against the wall of the cavity. Fresh and typical villi were found only at the site of the rupture. Although the author found in different parts of the wall some degenerated trophoblastic cells, he does not appear to have found even one spot where a typical chorionic villus, covered by its two layers—the Langhans' and the syncitial cells—burrowed its way into the ovarian stroma for sustenance. This, it would seem, is the important point. Furthermore, the Fallopian tube on that side was not examined microscopically. This case can, therefore, hardly be accepted as one of proved ovarian pregnancy.

Webster (1904).—This report is written so concisely and clearly that it will pay anyone to read the original article.

The case, however, may not be one of true ovarian pregnancy for the following reasons: 1. The fetus was not found. The operation was done about three months after the last menstruation and, therefore, the embryo must have been large enough to be visible. 2. The supposed amniotic cavity, well inside of the right ovary, contained only a few drops of turbid fluid. The wall is unbroken and does not show any rupture; nevertheless, about a half ounce of dark fluid blood was found in the utero-vesical pouch. This blood was not accounted for. Some have supposed that it came from the Fallopian tube on that side. It will be remembered that the tube on that side was not examined microscopically and, therefore, cannot be said to have been absolutely normal. With these important links missing, the chain of evidence cannot be said to be complete. One is, therefore, reluctantly compelled to place this case in that large, though unsatisfactory, class in which there is a probability that ovarian pregnancy existed.

Freund and Thome (1906).—Woman, 20 years old, who had always suffered from irregular menstruation, considered herself pregnant because of a three months' amenorrhea. The patient now suffered from continual discharge of blood from the vagina, also abdominal pain and faintness. Her abdomen enlarged and her physician, suspecting extra-uterine pregnancy, transferred her to the Strassburger School of Midwifery. Upon examination the following points were noticed: Abdominal tenderness; brownish red discharge from the vagina; uterus slightly enlarged, hard and movable. To the right and behind the uterus, a circumscribed, oval, fist-sized tumor of soft consistency was made out. At the laparotomy a persistent urachus and signs of infantile convolution of the tubes were seen. In the pelvis about 8 cc. of sero-

sanguinolent fluid mixed with remains of old blood clot and a gelatinous exudate were noted. Both tubes and the left ovary were normal and free from adhesions. The fimbriated extremity of the right tube was free and beautifully preserved, situated about 3 cm. from the growth and not connected with it. The growth was attached to the ovarian ligament. No perforation or rupture of the tumor wall could be seen. The growth and adnexa were removed. The patient recovered.

From the voluminous pathological report the following is abstracted: The tumor contains an amniotic cavity, 32 mm. in diameter, and is lined by the amniotic sac. The walls are from 1.2 cm. to 1.5 cm. thick, but at the site of the placental insertion the thickness is 2 cm. Within the cavity a fetus is visible, attached to the wall by a slightly twisted cord 11 mm. long. The embryo is of a dirty grey color, and its measurements are: length of body from neck to breech, 13 mm.; length of head, 10.5 mm.; thickness of head, 7 mm. Distinct and recognizable ovarian tissue was seen in the wall of the sac, but in no part were chorionic villi seen within the ovarian stroma. At a considerable distance from the placental insertion, almost invisible, striated, tubular formation can be made out. These do not take the stains well and may be interpreted as blighted chorionic villi.

It is a cause of genuine regret that the fetus was not sectioned to prove that it was, in reality, an embryo. It has been suggested that unless such proof were brought in these cases, some doubt may arise on that point. Leaving this question out of the discussion, one very important proof of the ovarian origin may be said to have been omitted, namely, the organic connection between embryo and ovary. This histological proof must be insisted on in all cases claiming the title of ovarian pregnancy. It is, furthermore, insufficient in this connection to mention the occurrence of some ill-defined, unrecognizable tubular formations, at a considerable distance from the placental site, and to suggest that these may be blighted villi. At the age of this embryo the development of the villi has so far advanced that they contain bloodvessels of their own. It is, therefore, reasonable to expect some explanation from the authors as to this sudden blighting of growing villi and their conversion into almost invisible, structureless masses. It is a recognized principle in the rules governing evidence, that the burden of proof rests on the claimant. Therefore, unless these points are cleared up, it is difficult to see how this case could be accepted as one of proved ovarian pregnancy.

Hewetson and Lloyd (1906).—A woman, aged 35, was admitted to the hospital as a case of subacute appendicitis. She had borne two

healthy children. The youngest of these was 18 months old. Her period was overdue by two weeks. She had been suffering from abdominal cramps and hemorrhage, and had passed some shreds and fragments of tissue per vaginam. Had vomited. On bimanual examination a doughy mass was made out, and also a slight resistance to palpation in the right iliac fossa. Temperature was normal. Pulse, 92; respiration, 24 per minute. On opening the abdomen about a pint of " porter-like " fluid was evacuated, and in the deepest part of the pelvis a mass of firm blood clot, the size of a fist, was seen. This was removed. The uterus was slightly enlarged. The right tube was free, as was also its fimbriated extremity. The right ovary was enlarged, and formed part of the wall in which the above-described blood clot had lain. A ragged opening was seen in the posterior part of the ovary. Evidently, this was the site of the bleeding. The left adnexa were quite normal. The right ovary and tube were removed. The patient recovered. The fetus was not seen, not even some part of the chorion or amnion. Sections of the tube showed it to be normal. The blood clot showed clusters of chorionic villi scattered irregularly through it. Villi were visible also in the gestation-sac. At no point do the villi penetrate to the ovarian tissue.

With no fetus seen and no evidence that it ever was connected with the right ovary, at least in the intimate relationship which exists between ovum and maternal tissue of its implantation; with no proof of ovarian structure in the gestation-sac, and, above all, no villi eating their way into the ovarian stroma, one would find it difficult to accept this as a case of true primary ovarian pregnancy.

Law (1906).—A nullipara, aged 30, had always been regular in her menstrual periods. The last period took ten days—three more than usual. The patient was subject to attacks of biliary colic. The lower half of the abdomen was tender. No mass palpable. Bimanual examination impossible. No vaginal discharge. Temperature, 101° F. Pulse, 120. At the laparotomy about one and a half pints of dark blood welled up out of the pelvis, and deep down it was filled with recent clot. The source of the bleeding was seen to be a ragged cavity in the left ovary. This, together with its tube, was removed. The other pelvic organs were normal. The patient recovered.

The specimen was sent for examination to the " Clinical Research Society " and an abbreviated report is here added:

"This ragged cavity is a gestation sac. The wall is formed of ovarian tissue lined with fibrin and blood clot, in which are imbedded several chorionic villi and masses of fetal epithelium. As the corresponding tube is free of the gestation-sac, it is very probable that the specimen represents an ovarian pregnancy. However, it is too much damaged for a positive opinion on this

point." The last sentence of this report may be accepted as the summary. The case can, therefore, not be regarded as one of proved ovarian pregnancy.

KELLY AND MCILROY (1906).—V-para, aged 33 years. The last child was born two and a half years before admission. All puerperia were normal. Menstrual period delayed two weeks. She was seized with a sudden attack of pain in both iliac regions. No vomiting. No tumor palpable. Uterus movable, and slightly enlarged. The left appendage forms an enlarged and tender mass. The right appendage is tender, but shows no enlargement. She has been bleeding from the uterus irregularly for about a month. She was treated by the expectant plan for ten days. Laparotomy was performed almost three months after her last menstruation. A blood cyst, the size of a large plum, was connected with the left ovary. The left ovary and tube were removed. The right adnexa were normal. A few months later the specimen was examined. On cutting the tumor no embryo was found. Traces of chorion, amnion and villi were found in the central portion of the blood mass. The upper part of the specimen is undoubtedly ovarian tissue. Nowhere can villi be seen in direct contact with ovarian stroma.

As the fetus was not seen nor its place of nidation proved to have been in the ovary, and not even one villus traced into ovarian tissue to prove that the ovum actually got its sustenance from this organ, one can hardly agree with the authors of this report that the case was true ovarian pregnancy.

M. CHENOT (*Gynecologie*, December, 1906, p. 481).—Woman, 29 years of age, entered the hospital Nov. 13, 1906. She showed symptoms of ruptured ectopic. Her menstruation was delayed several days. She menstruated regularly Sept. 16, 1906. As she did not see the next period, she took a large amount of apiol. On October 20, she got violent abdominal pains. Near the end of October she had metrorrhagia and passed a big clot and fainted. The bleeding stopped and she remained well until November 13, when she entered the hospital. She presented all the symptoms of collapse, and on palpation no tumor in the abdomen could be made out. The breast contained colostrum. On laparotomy a dermoid cyst was found in the left side. A ruptured ectopic was found on the right side. This was removed. The woman recovered. The histological examination of the specimen will be published later. The tube is greatly thickened and the fimbriæ hardly visible.

No mention of a fetus. Only an empty sac adherent to the apparently pathological tube.

This case cannot be considered an ovarian pregnancy.

HERRENSCHMIDT AND RIGOLLOT SIMONOT (*Bull. de la Societe Anatomique de Paris*, 1906, LXXXI, p. 289).—Woman, 25 years old; nullipara. Last regular period, Jan. 27, 1906, lasted three days. On Feb-

ruary 10 some abdominal pain; next day she again experienced this pain, dizziness and faintness and had cold perspiration on her forehead. As the symptoms grew alarming, she went to the Necker Hospital. She showed signs of internal hemorrhage. At laparotomy about a liter of fluid blood, also clots, were seen. The right tube of normal aspect; the ovary not enlarged, but on its surface is a small ulceration from which blood is constantly oozing. On the left side the adnexa are enlarged; the ovary is cystic, but there is here no hemorrhage. During the manipulation incident to the ablation of the right ovary, a bean-sized body, resembling a blood clot, is expelled from this ulcerated area on the ovary and lost in the abdominal cavity. The tube and ovary were removed. Peritoneum drained. Good recovery. The ovary is a little enlarged. On its free border is an ulceration, and at its edge is a clot. The cavity is circular in shape, 3 or 4 mm. in diameter, and from 2 to 3 mm. in depth. Several parallel sections made through the ovary demonstrate the presence of some corpora albicantia. No corpora lutea are visible. At the microscopical examination it was seen that ovarian tissue was present in the walls of the cavity. On the inner side lutein cells are visible in large numbers. This field of lutein cells is divided into irregular spaces by bundles of vessels. No trace of infiltration with blood. In the bottom of the cavity is a conglomeration of cells and fibrin. Chorionic villi are to be seen in the walls of the cavity. Fetus not found. Tube not examined.

Comment on this case is almost superfluous, except that it cannot be accepted as a case of proved ovarian pregnancy.

Shickele (*Hegar's Beitraege z. Geburts and Gyn.*, XI, 1907).—IV-para, 28 years old. After her menses had been absent about seven weeks, she bled profusely for several days, during which time shreds and a piece of skin were passed. For the next two weeks she had pains in the lower abdomen. She bled again for three days. She never had any symptoms of faintness or collapse. On admission to the hospital a diagnosis of left-sided tubal abortion with hematocele was made.

On opening the abdomen a few coils of small intestine are seen to be adherent to the crest of a soft, dark blue sac, which lies behind the uterus and appears to completely fill the true pelvis. On the left side, the normal-appearing tube is seen on top of the sac, passing along in an undulating manner. At the right edge of the sac the normal right tube and ovary are visible. The attempt is made to loosen the sac from its light attachments to the wall of the pelvic peritoneum. In lifting it to the abdominal incision, the sac bursts and a considerable quantity of old blood coagula escape. The sac is now brought forward and with the left tube and left ovary, which lies above it, clamped and removed. Vaginal drainage. Closure of abdomen. The woman recovered.

At no point can chorionic villi be found within the *blood-mole*. On the contrary, the villi are free within the blood masses No trace of decidual formation is evident anywhere. The corpus luteum is filled with sero-sanguinolent fluid; no connection exists between this

corpus luteum and the blood mole. Implantation did not by any means take place within a follicle.

The last sentence may be considered the verdict in this case, namely: ovarian pregnancy is positively excluded.

Michaux (*Bull. et Memoirs Soc. de Chir. de Par.*, 1907; *n.s.*, XXXIII, p. 560).—A case of extra-uterine ovarian pregnancy.

Michaux exhibited a specimen (removed by laparotomy on that day) to the Surgical Society. This he modestly named extra-uterine pregnancy. It shows the thickened tube entirely closed at the orifice, and an enlarged ovary from which a mass of yellow fluid escaped at the time of removal. This semi-fluid, yellow mass was about the size of a large raisin and contained a small, pink body which he believed to be an embryo. He does not think he could be mistaken. However, the specimen will be examined microscopically.

Without the microscopical report one cannot say whether this is a case of ovarian pregnancy or not.

History.

The patient, Lillie M., of Bath Beach, L. I., a well-nourished and healthy looking woman of medium size, presented herself at the office on November 21, 1906, and gave the following history:

Age, 28 years; occupation, housewife; married eight years; family history unimportant; never had any serious illness or injury. About two years before marriage she received a blow on the small of the back while in a garden swing, and since that time she has suffered some pain at each menstrual period. First menstruation at 14 years. Since then the periods have recurred every 30 days, except during pregnancy or lactation. When married seven months her uterus was divulsed and curetted for tight os. Two years and three months later she gave birth to a child at term, without artificial aid. The child is living and in good health. While nursing this child, which was then seven months old, she miscarried at three months. Was curetted. In November, 1905, she again miscarried and was once more curetted. This time she was pregnant four months. The last regular menstruation began September, 1906, and lasted the usual time. It was due on October, 12, but failed to appear. Four days later, viz., October 16, she found herself "spotting." This continued for four days, when she got cramps and passed a piece of "flesh" about the size of a hazel-nut. This was on the 20th—eight days after the date on which she missed her

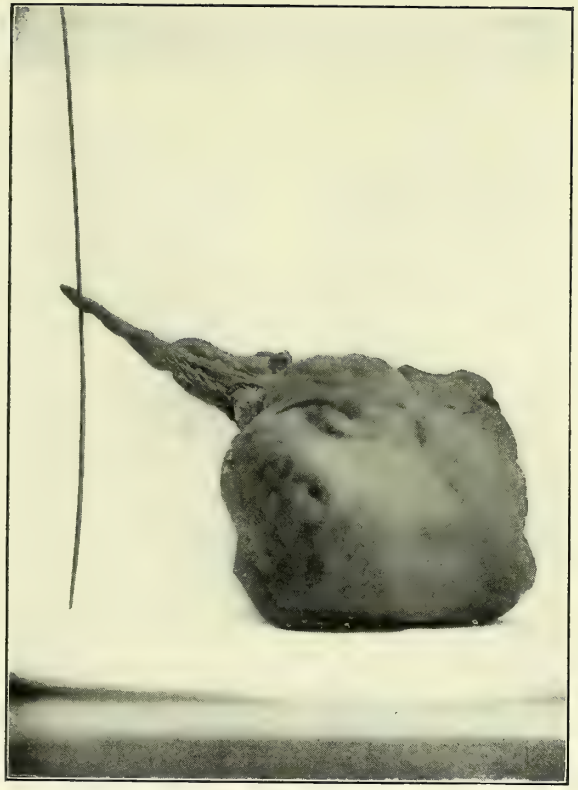


FIG. 1.—Ovary and tube, reduced in size, front view. Tube rotated on its axis by mistake.

(Jacobson)

FIG. 2.—Ovary and tube, back view, reduced in size, showing fibriated extremity quite normal and non-adherent to the ovary.

menstrual period. She came to me on the following day for an opinion.

Physical examination showed the heart, lungs, liver, and kidneys to be normal. There was no rigidity of the abdominal muscles nor was anything abnormal to be felt within the abdomen. *Vaginal examination* disclosed a gaping introitus stained with fresh blood. Bimanually, the cervix was found to be softened and slightly patulous. The uterus was somewhat enlarged, in good position and not specially tender. The right adnexa were apparently normal. The left Fallopian tube appeared to be quite normal. Occupying the place of the left ovary was a round, smooth, elastic, freely movable swelling which gave the impression of being the left ovary enlarged to perhaps four or five times its normal size. In view of the history and findings, a diagnosis of extrauterine pregnancy was made. The tentative diagnosis was probable tuboovarian pregnancy. Immediate operation was advised. The patient thereupon was admitted to the Gynecological division of the Sydenham Hospital.

After the preparation of the patient for laparotomy had been carried out in the usual manner, operation was performed the following day.

Operation.

A median incision about four inches long was made through the linea alba. At this point it was noticed that the abdominal walls were extraordinarily vascular. This tended to confirm the diagnosis of ectopic pregnancy. It has been the writer's experience that in operations for extrauterine pregnancy the abdominal walls are very vascular and bleed excessively. The peritoneum having been divided the full length of the incision, the patient was placed in the Trendelenburg position, a large sized broad retractor placed in the lower angle of the wound, and the intestines packed away by a large abdominal sponge. On introducing the hand within the pelvis it was noticed that the uterus was but slightly enlarged, in good position and freely moveable. Running the fingers along the left Fallopian tube, the latter appeared to be normal in every way; and thus the left ovary was reached, which seemed to be considerably enlarged, but not adherent. A heavy chromic-gut ligature was passed around the ligamentum infundibulo-pelvicum and securely tied,

cutting off the blood supply through the ovarian artery. A similar ligature was passed around the Fallopian tube, near the uterine cornu, and tied. This controlled the supply from the uterine artery. Finally, another ligature was passed around the ovarian ligament and tied. The ovary, also the part of tube and ovarian ligament situated between the ligatures, were cut away with scissors and removed. A running catgut suture united the cut ends of the broad ligament, leaving no raw surface exposed. The opposite ovary and tube were drawn out of the wound for inspection. They appeared to be normal and were returned to the abdomen. After inspection of the pelvis, to be sure that there was no oozing, the abdomen was closed by tier sutures. The patient made an uneventful recovery. At this writing, 15 months after the operation, she is enjoying the best of health.

Pathological Report.

The specimen — obviously the left ovary — was irregularly spherical in shape and slightly flattened from before backwards. It was about 8 x 7 x 6 cm. in size and weighed about 36 gm. Its surface was smooth and glistening and mottled in fairly large patches, the color varying from bluish gray to burgundy-red. On the postero-superior border of its proximal surface a short bundle of connective tissue fibres marked the place of attachment of the ovarian ligament. On the opposite or external border the left Fallopian tube was attached by well-marked ovarian fimbriæ. The tube, with its fimbriated extremity in apposition to the ovary, but not adherent to the latter, was open and apparently normal in every way. It was about 8 cm. long. The organ was firm in consistency, except in one spot at the lower border, which was darker in color than the rest of the organ and noticeably softened to the touch. Fearing that the embryo, which was expected to be very small, might be lost in the blood clot and the specimen thus destroyed, it was decided not to follow the natural impulse of cutting the ovary open, but on the contrary to harden it first. Consequently, the specimen was placed into Pick's solution—a mixture of Formalin and artificial Carlsbad salt—for a week, to harden and also to retain and fix permanently its beautiful markings and coloring.

The ovary was then taken to the Post-Graduate Hospital Laboratory and, in the presence of Professor Henry T. Brooks

[Fig. 4.—Ovary cut through at a low level, showing the tiny embryo in situ.
(Jacobson)

Fig. 3—Ovary cut open (slightly reduced).

and assistant Professor Benj. Cline and Dr. T. Homer Coffin, cut longitudinally across. The two halves of the organ were separated, and it was seen that the parenchyma was densely infiltrated with blood. About 4 mm. from the cut edge, and extending well towards the centre of the organ, was a small cavity containing a few (perhaps five) drops of limpid fluid. Floating in this fluid was a tiny, white embryo which was attached to the inner surface of the cavity by a slightly twisted, transparent membrane. This membrane arose from the caudal extremity of the embryo and, before reaching the point of its attachment, it spread out mushroom-shape so as to obtain a broad base of insertion. It was fortunate that the knife missed the embryo and merely cut through the amniotic cavity. The specimen, therefore, justified the diagnosis previously made and represented a very early stage of an exceedingly rare condition, namely, *true primary ovarian pregnancy.* In this opinion the following gentlemen also concurred: Professor H. J. Boldt; Professor George M. Edebohls; Professor Joseph B. Cooke; Professor Jeffries, Pathologist to the New York Polyclinic; Dr. Edward L. Oatman, Pathologist to the Manhattan Eye and Ear Hospital, and Dr. George E. McLaughlin, Pathologist to Christ Hospital, Jersey City. There were many others quite competent to judge who also were of the opinion that this was a case of true ovarian pregnancy, but I shall refrain from swelling the list.

The embryo measured 6mm. in length and 2 mm. in thickness. On viewing it through a magnifying glass, a head, rudimentary eye, branchial clefts, curved back and pointed lower extremity, were seen. A rudimentary cord or membrane attached the embryo to the ovary and four buds marked the site of the two upper and two lower extremities.

For microscopical study, coronal and sagittal sections were cut from the specimen, each about 1 cm. thick, and these were dehydrated and imbedded in celloidin in the usual manner. The microscopical sections were stained by the hematoxylin-eosin method, and mounted in Canada balsam.

Upon examination the most striking feature was the intense infiltration of the tissues with blood. This was so decided in whole areas as to make the impression of blood clot. In almost every field chorionic villi, large and small, were to be seen, giving classical pictures of the two epithelial layers—the Langhans'

and the sincytial layer—the lumen showing branched connective tissue cells that resembled myxomatous tissue. In some sections this picture of blood clot perforated by villi dominated the whole field and only around the edges could ovarian tissue be found on close inspection. Sections of the central portion showed a rounded cavity which had contained the embryo. The cavity was lined all around its inner margin with a narrow strip of tissue consisting of two rows of cells of chorionic origin. This cavity could not be demonstrated in any part to communicate with the surface of the ovary but, on the contrary, at the place where it was peripheral there was a bridge of tissue from 3 to 5 mm. thick between it and the surface of the organ. This proved the cavity to be situated well within the ovary, and not an artefact in the sense that it was originally formed upon the surface. That true and definite ovarian tissue comprised the zones bordering this cavity, in places where blood infiltration did not obscure the picture, the following elements of normal ovarian parenchyma demonstrated:

1. Arterial and venous capillaries, some with and some without blood within the lumena.

2. Whorls and eddies of spindle-shaped cells arranged concentrically around blood spaces and sometimes independent of these.

3. A corpus albicans.

4. Graafian follicles of various sizes.

5. Towards the hilum, thick-walled, cork-screw-shaped vessels which are usually found in this region.

6. Condensation of the cortical tissues near the surface, erroneously called tunica albuginea, was quite apparent.

Clusters of round cells were found occasionally, and also some large wandering cells so frequently seen on microscopical examination of the pregnant uterus.

The fetus was sectioned sagitally and shows umbilical cord with the lumina of two vessels in it. It is attached organically by this cord to the cavity within the ovary.

Chorionic villi are seen eating their way into the ovarian tissue.

Summary.

Briefly, the reasons given by the writer of this paper for believing his case to be one of true primary ovarian pregnancy are as follows:

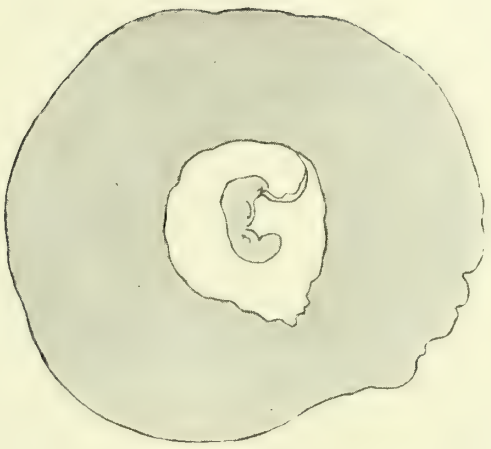


Fig. 5—Schematic drawing of embryo in situ (slightly enlarged).

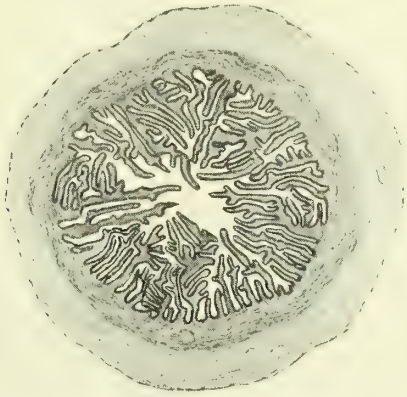


Fig. 8.—Normal Fallopian tube of side corresponding to impregnated ovary. (Jacobson)

1. This patient, a woman of the childbearing age, "skips" a menstrual period. A few days later she suffers from abdominal cramps and has a bloody discharge from the vagina. She later passes a piece of "skin." The bleeding and cramps continue. On bimanual examination the place of the left ovary is occupied by a smooth, rounded, noninflammatory mass. The uterus is slightly enlarged and softened. Clinical diagnosis of ectopic pregnancy was irresistible.

2. At the operation the uterus was found to be but slightly enlarged, in good position, and freely moveable. The right ovary and tube were normal. The left tube was normal and nonadherent. No signs of any kind of inflammatory trouble were visible in the pelvis. The left ovary was enlarged but showed no pathological adhesions. The left ovary was attached to the uterus by the ovarian ligament.

3. After hardening, the ovary was bisected and showed almost in the centre a small cavity which did not communicate with the surface of the ovary. This little cavity contained a few drops of clear fluid. Within the cavity and suspended in the fluid, by means of a short, somewhat twisted cord, was a tiny human embryo. The cord was attached, on the one hand, to the lower portion of the ventral surface of the embryo and, on the other, it was attached to the lining of the cavity in which it was situated.

4. The embryo was examined microscopically by Dr. D. W. Cairns and was not only shown to be a very young fetus, but in addition a section through the fetus and its cord show how the latter is organically attached to the ovary. This proves that the fetus actually grew there.

5. Microscopical sections of the ovary show not only *all* the histological elements of ovarian tissue, thus proving that it is positively ovarian structure, but, furthermore, they show the fetal villi eating their way into the ovarian substance, which proves that this fetus received its sustenance and support from the ovary itself.

6. Sections of the left tube show it to be absolutely normal in every way. All these proofs of ovarian pregnancy having been demonstrated, and the claim to the title of true primary ovarian pregnancy having been established beyond a doubt, further investigation was abandoned.

In conclusion I wish to thank Professor H. T. Brooks, for valuable aid in the preparation of this paper and Dr. Douglas W. Cairns for cutting the embryo so beautifully.

Appendix.

It may, perhaps, not be amiss to touch lightly upon one of the most important and interesting phases of embryology, namely, fecundation. As this study is in line with the subject-matter of this paper, and in view of the fact that the writer is firmly of the opinion that, normally, impregnation of the human being always takes place in the ovary, a short digression into the field of speculation may perhaps be considered pardonable. On the other hand, the limits set to this paper having been reached, brevity has become imperative; therefore, no attempt at a scientific discussion will be made, nor authorities cited, but this more complete treatment of the subject deferred for a paper which it is hoped will be published shortly. The reasons which may be given for believing that impregnation takes place in the ovary are briefly as follows:

1. *Comparative Biology.*—In the lower and higher order of vertebrates, fertilization takes place by the male depositing its seminal fluid upon the ova immediately after their being laid by the female. For instance, in the case of some fishes, the female lays the ova (roe) in a protected, shallow pool; the male, swimming leisurely over this mass, drops its seminal fluid upon it. Thus, without contact of the male and female genitals, fecundation is established. The valuable lesson taught by this study is, that the essential act consists in a union of male and female elements and not, necessarily, contact of male and female genitalia.

a. The brilliant and successful experiments in artificial fecundation carried out by Spallanzani, Prevost and Dumas, and Rusconi.

b. Contrarywise, this is proved also by animal experiments—the socalled negative method. By preventing the union of spermatozoa and ova, impregnation was prevented. Eminent in this field were Haighton, Nack, Grossmeyer, Cruikshank and Blundell.

2. *Observations made on the human being.*—

a. Lecluyse reported a case of abdominal pregnancy which is

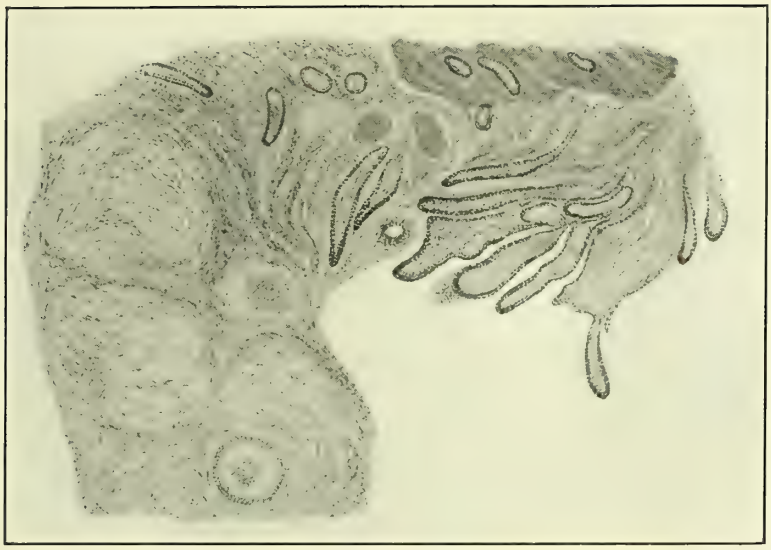


FIG. 7.—Portion of ovary at site of attachment of cord, showing fetal tissues organically connected with the ovary.

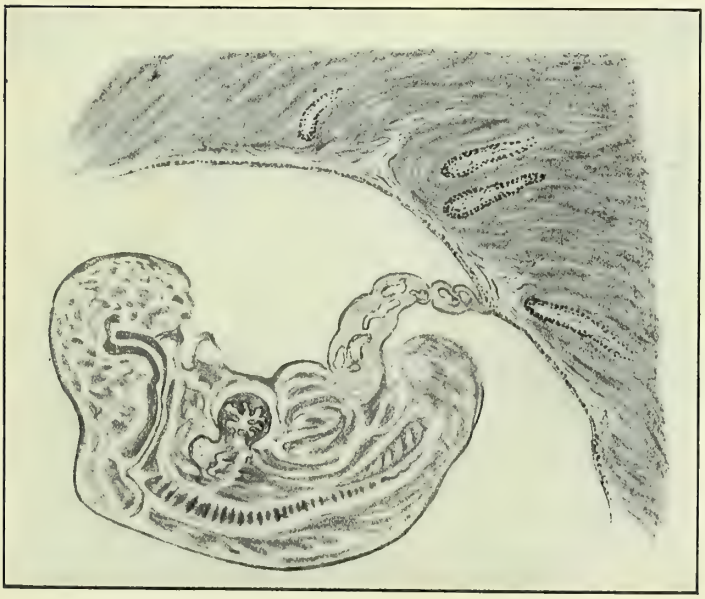


FIG. 6.—Embryo, enlarged, Longitudinal section through embryo and funis, showing attachment to the ovarian structure by vascular cord. (Jacobson)

most extraordinary and interesting. A woman had gone through a Cesarean section for contracted pelvis. She again became pregnant, and giving evidence of internal hemorrhage, he performed laparotomy. The patient died. It was found that in the line of the uterine scar left from the previous section, a fistula, 4 cm. long, existed. This made a broad communication between the cavity of the uterus and the pelvis. It may be assumed with the author that through this fistula the impregnated ovum wandered into the abdomen and implanted itself there secondarily. Of this there was no conclusive proof. Or it may be assumed that the uterus had never been the place of nidation of the ovum, but that it settled primarily in the abdomen, and was one of the instances of primary abdominal pregnancy.

In this case the Fallopian tube was not necessarily the passage for ovum or spermatozoon.

b. Koeberle extirpated a fibroid uterus. It was evidently a supravaginal hysterectomy. There remained a fistula in the scarred cervix. Through this fistula the patient conceived again, and of necessity the pregnancy was now extrauterine. This ruptured and the patient died. One may, therefore, conclude that, for impregnation, a uterus is by no means necessary.

There is not one authentic instance on record where there was no ovary, and yet impregnation took place. Even a small part of an ovary, if capable of functionating, is sufficient.

3. *Anatomical reason.*—The Graafian follicle in the human ovary has, briefly speaking, the following structure. It consists of an external fibrovascular coat, connected with the surrounding stroma of the ovary by a network of bloodvessels. It also exhibits an internal coat, named ovicapsule, which is lined by a layer of nucleated cells, named the membrana granulosa. The fluid contained in the follicle is transparent and albuminous. In that part of the Graafian follicle *which is towards the surface of the ovary*, the cells of the membrana granulosa are packed together into a heap, and this heap is named the *discus proligerus*. In this discus proligerus the ovum lies imbedded.

a. All intraperitoneal organs are covered by peritoneum. The surface of these organs is covered by flat epithelial cells. The ovary is the only exception. Its covering consists of *columnar cells*.

b. During the process of maturation the Graafian follicle approaches constantly nearer to the surface of the ovary; its fluid contents increase rapidly, causing distention of the walls; the follicle, still enlarging, bulges above the niveau of the organ; its covering thins out, more and more, and becomes so attenuated that one can easily see the shimmer of the follicular contents through it. Finally it can not longer stand the constantly increasing pressure from within and the thinning of its walls, a defect is caused and the follicle ruptures.

Let us suppose for a moment that a spermatozoon, having traveled up the uterus and Fallopian tube, has reached the surface of the ovary. (Bischof has seen the spermatozoa swarming on the surface of a rabbit's ovary). It would not be impossible for such a spermatozoon to penetrate through the extremely attenuated wall of a mature follicle and, finding the ovum securely imbedded in the discus proligerus bulging out over the surface of the ovary, gain access to the ovum and unite with it in the way taught by all text-books on the subject. This entrance would be facilitated by the anatomical arrangement of the columnar cells upon the surface of the ovary. It is a simpler matter to force a way through the comparitively soft cement substance *between* two columnar cells than to be compelled to *bore through* a flat epithelial cell. The difference might be likened to driving a nail into a brick wall *between* the bricks or *through* a brick. The ovum, being held fast, could not recoil from the attack, and almost all mechanical advantages being in favor of the spermatozoon, it would be almost certain to win.

4. *Mechanical Reason.*—For the purposes of this paper the ovum may be considered a ball and the spermatozoon a wedge pointed stick. Once the Graafian follicle has burst and the ovum is set free, the latter is precipitated into a comparatively large space—the peritoneal cavity. Here it rolls around, driven in various directions by the ceaseless motion of the pelvic and abdominal viscera. It is finally aspirated, together with some serum and perhaps a few drops of blood, into the abdominal ostium of the Fallopian tube and slowly approaches the uterine cavity. It eventually drops into the latter and is discharged and lost.

Now let us see for a moment what would probably happen if

impregnation took place anywhere else except in the follicle. The spermatozoon meets the recently extruded ovum. It attacks the latter. At the first impact the little globe would roll along. Much the same thing would occur as when a ball is poked by a stick. The mechanical disadvantage under which the spermatozoon would be placed might well frustrate every attempt at union. The mechanical disadvantage would be heightened by the fact that the little globe is covered with secretion from the tube, lying on a slippery surface, in unstable equilibrium, and the peristaltic motion of the tube towards the uterus would complete a chain of unfavorable circumstances which, even the indomitable little spermatozoon, may not overcome.

5. *Physiological Reason.*—In the wonderful scheme of nature, things are not done in a haphazard way. It is the manifest object of the spermatozoon, be it human or animal, to wriggle its way up to the ovary. These little beings have been found by different observers in every part of the genital tract. Most significant of all observations, spermatozoa have been seen repeatedly swarming on the surface of the ovary after copulation. Was that merely accidental? Does it not rather give us a hint of the scheme of creation? If the spermatozoon could just as easily unite with the ovum in the lower portion of the genital tract, would it not stay there and wait for the inevitable arrival of the latter from above?

Although the theory of normal impregnation taking place in the Graafian follicle may not be generally accepted, the writer's case proves that impregnation can occur in the ovary.

616 Madison Ave., New York.

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REPORT OF A CASE OF ATRESIA OF THE VAGINA WITH HEMATOMETRA, HEMATOSALPINX AND HEMATOVARIUM.

Including a Resumé of the Literature of the Blood-Retention Tumors, Published During the Past Twenty Years.

BY ABRAM BROTHERS, B.S., M.D.

Adjunct-Professor of Gynecology, New York Post-Graduate Medical School and Hospital.

On Nov. 26, 1907, Dr. H. Lewinsohn invited me to examine Miss L. A., who gave the following history. Although 21 years old, she had never menstruated. Since she was 17 years old she had been troubled with a dull pain in the lower abdominal region. Every four weeks the abdominal pain became quite severe and sharp in character during five days. Her last menstrual crisis was rather less painful than usual. For some time past she had noticed a gradual enlargement of her abdomen. There was nothing of importance which could be elicited in her previous history or in that of her family.

The girl presented a fairly well nourished appearance, although anemic. The breasts were well developed, but there seemed to be an increased growth of hair on the body and limbs. Inspection of the abdomen showed a protrusion which, on palpation, could be clearly mapped out as filling up the entire right side and extending half way over to the left. Its upper margin extended well up above the umbilical region and presented a marked sulcus just about the navel. The tumor gave a sense of fluctuation, on manipulation, and a flat note on percussion. Locally, the external genitals presented fairly normal appearances. The *mons veneris* and *labia majora* were perfectly normal. The clitoris was small and its prepuce passed on continuously into the *labia minora* which, however, seemed to extend downward only a short distance. The entire interlabial space was occupied by a longitudinal slit, about $\frac{1}{2}$ inch long, which was recognized as the urethral orifice. Below this a small fraction of an inch of dense reddish tissue was continuous with the perineal skin beyond. There was nothing to suggest the presence of a hymen

or a vagina. The little finger was easily passed into a capacious bladder and was able to feel the fluctuating tumor beyond. Replacing the finger with a catheter and passing the index finger into the rectum, a thin partition was felt which represented the site of the vagina. Nothing like a cord was palpable. At a depth of two inches something hard was recognized as the cervix. Beyond this point it seemed to merge into the tumors above. The diagnosis was made of absent vagina with retention of blood in the uterus and right adnexa.

Three days later under anesthesia the patient was re-examined. The large, right-sided abdominal tumor was found to be round in shape and to extend well up to the liver region. Its upper contour curved downward in the median line where, below the umbilicus, it became continuous with a second tumor which, by recto-abdominal palpation, was recognized to be an enlarged uterus. The partition between the bladder and rectum was re-examined and estimated to be, at the utmost, one-eighth of an inch in thickness. The inadvisability of attempting to do anything from below was quite apparent.

A median longitudinal incision—about five inches long—was made through the thin abdominal wall and a diastasis of the recti muscles in the upper portion of the wound was noted. On opening into the peritoneal cavity a large round tumor, the size of an adult head, was seen presenting the usual aspect of an ordinary large unilocular ovarian cyst. The urachus was recognized to the left of the incision and followed downward in the abdominal wall to its attachment, below the brim of the pelvis, in the bladder. In the left pelvis the exploring finger discovered a distended tube with one or two compartments and adherent to an orange-sized ovary. Several peritoneal blebs and adhesions about this tube and ovary indicated the existence of recent and old pelveo-peritonitis. There was no free blood in the peritoneal cavity. The uterus was larger than normal, felt boggy to the finger and reached well up to the level of the umbilicus. The right elongated Fallopian tube at its uterine end presented the thickness of the little finger. It crossed in front of the large right-sided tumor, from which it was at first fairly distinct, to its right side and was lost behind in the tumor wall with which it became inseparably fused. Both round ligaments, although somewhat thickened, occupied their normal anatomical positions.

Behind the large, right-sided, globular tumor a cord-like structure, as thick as the little finger, prevented its delivery from the wound. Otherwise the adhesions on this side were few and only in the lower field of operation.

Dividing the large band alluded to—and which passed between the posterior surface of the tumor and the posterior parietal peritoneum—it was possible partially to deliver the tumor. In separating adhesions low down and behind, a rupture of its wall took place at one point which, before it could be caught in a clamp, permitted the escape of possibly a half pint of chocolate-colored liquid without clots into the abdominal cavity. The tumor—still containing about the same quantity of liquid—was rapidly caught at its pedicle with clamps (which were later replaced by ligatures) and cut away. The abdomen was now carefully cleansed with gauze sponges and another inspection of the conditions present was made. The appearance of the left adnexa justified complete extirpation. To leave the uterus might have been feasible, but would have implied (1), either trusting to nature to absorb a rather large quantity of blood in its interior, which might at any time undergo suppuration; (2) or incising the uterus, wiping out its contents, and either sewing up the uterine wound or draining it through the abdominal incision; or (3) attempting to reach the cervix and uterine cavity by digging a passage-way through the occluded vagina. Neither of these plans appealed to me in this individual case, and I reasoned, in addition, that, as the adnexa had under all circumstances to be sacrificed, there was no object in subjecting the patient to possible further dangers by the retention of an organ which was now of no use whatsoever. Hence, I proceeded to remove the uterus and opposite adnexa in one mass.

Securing the vessels on each side in the customary manner, and dividing the peritoneum anterior and posterior to the uterus, the cervix was dislodged from its surroundings by means of the scissors and found to open into a small, upper rudimentary vagina. The cervix was found distended with blood and as large as the average uterus. The vaginal cul-de-sac, about ½ inch in depth, was seen to contain a small quantity of tarry liquid. The same thick, stringy, tarry material oozed out of the cervical and uterine cavities. Cutting the cervix from its surroundings, the uterus and left adnexa were removed in one mass.

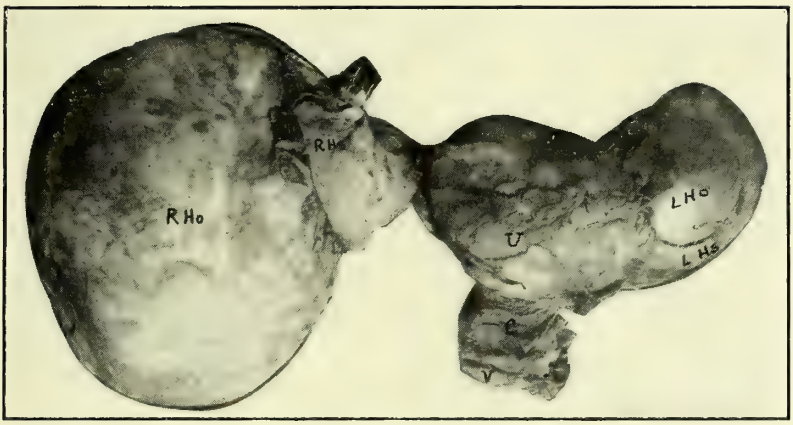

FIG. 1.—Anterior aspect. RHo, right hematovarium; RHs, right hematosalpinx; U, uterus; LHo, left hematovarium; LHs, left hematosalpinx; C, cervix; V, fragment of vagina. (The right Fallopian tube is tilted to show patent, but not distended, uterine portion.)

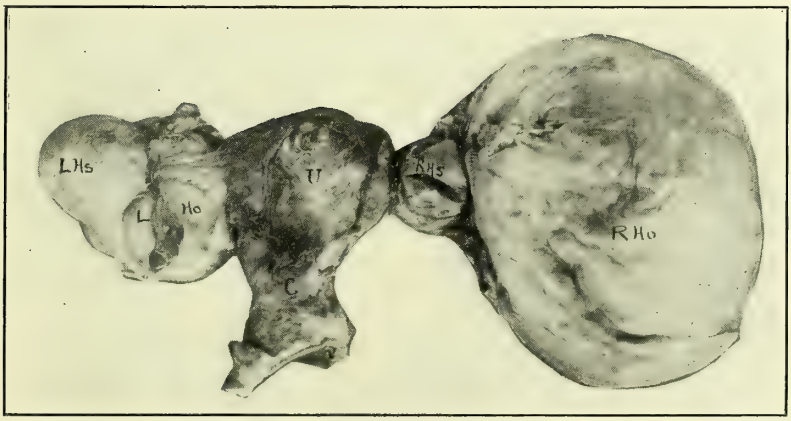

FIG. 2.—Posterior aspect. (Brothers)

The amount of bleeding from the operation—which presented considerable technical difficulties and consumed nearly an hour and a half in its performance—was *nil*. The question of constructing a new vagina with the rudimentary upper portion serving as a guide was dismissed, as I feared the effect of prolonging the operation unnecessarily. The edges of the vaginal stump were now inverted—after wiping its cavity thoroughly dry—and sutured to each other, and over this the peritoneum was sewed. After making sure that the peritoneal cavity was thoroughly cleaned with dry sponges, the abdominal wound was closed in three layers without drainage. There was no reaction and, at the end of a week, the patient was sitting out of bed. Several months later her physician reported to me that she was perfectly well.

The specimens, which were presented at the February meeting of the New York Obstetrical Society, have shrunken so much since the operation that it requires a stretch of the imagination to recognize them. Briefly described, they consist of a thick-walled uterus and cervix to which a fraction of vagina is attached below like a cuff. The right tube is pervious at its uterine end. The ovary on the right side is one large, globular mono-cyst, the dimensions of which had shrunken, probably, by at least one-half at the time the photograph was taken. The posterior aspect of the right tube shows a very thin wall—as though rupture might have occurred at any moment. The left tube, in the posterior view, is seen to end in a club-shaped dilatation which was fused to the left hematovarium. The left ovary—which like its tube—is a single sac filled with blood, shows numerous adhesions on its posterior aspect. The left tube embraces the corresponding ovary from below upwards for a distance of two-thirds of its circumference.

The points of interest in the case are the following: (1) the absence of nearly the entire vagina with normally developed functionating uterus and appendages; (2) the accumulation of blood during a period of four years in the rudimentary vagina, cervix, uterus, tubes and ovaries without rupture; (3) the mono-cystic hematovarium, which, I believe, is the largest one on record. Let us consider each of these subjects in turn.

According to Dohrn, the union of the Müllerian ducts in the human embryo is completed by the ninth week, but the differen-

tiation of the genital canal into uterus and vagina does not occur until much later. Kussmaul places it at the end of the third month, although Dohrn could not locate the cervix until the fifteenth or sixteenth week. The lower edge of the tube, which later is the site of the hymen, is situated and opens into the sinus-urogenitalis. The hymen makes its appearance about the beginning of the nineteenth week and is originally a simple projection at the posterior wall of the introitus. This is the normal condition in the embryo; so that by the fourth or fifth intra-uterine month the genitals have reached their completed development (Breisky).

Nagel, using the above embryological data as a basis, made the claim that congenital absence of the vagina could not occur with complete development of uterus, tubes and ovaries; that where such occlusions exist, they are the result of a membranous formation which takes place *after* the completed development of the vagina in the later months of pregnancy or after birth; that it is the result of adhesion of opposite vaginal folds and due to the absence of the superficial layer of epithelium; and that such tissues may contain connective-tissue, muscle fibres and blood-vessels, because the vaginal folds are usually supplied to the very periphery with blood-vessels. Hence, he concluded that these cases must always be the result of *acquired atresia.*

Veit went further. He claimed that the presence of an hematosalpinx in such cases was proof of infection in the genital tract and, hence, reasoning backwards, that the atresiæ were necessarily acquired and not congenital. He claimed, in addition, that not only was this the case in the single genital canal, but that in cases of blood-retention in the double genital canal, the atresia was not necessarily congenital and, when with associated hematosalpinx, must be always acquired.

The Nagel-Veit theory received wide recognition throughout the medical world by the leading writers on gynecology—especially in Germany. Thus Robert Meyer, Breisky, Zweifel, Gebhard, Pick, Pincus and a host of others accepted the theory. Pincus, in fact, wrote an exceedingly voluminous and interesting monograph on the " Practical and Important Questions Dependent on the Nagel-Veit Theory " (*Sam. Klin. Vorträge*, 1901, p. 219). Within recent years there has been considerable opposi-

tion from Sänger, Stratz and others. Labusquiere, after a searching criticism of the subject, comes to the conclusion that "the proposition of Nagel, Veit, Meyer, according to which a hematosalpinx is the criterion of a previous infection, is not sustained —it is false."

Döderlein and Krönig, in their text-book on gynecology, just published, make the most serious attack on the theory. We translate freely: "As clinicians, we are not friendly disposed to the Veit-Nagel theory. We rather agree with Mainzer, because two segments of the Müllerian ducts are recognized in the lining epithelium of which the lower (which has no cavity and is filled with large four-cornered cells) becomes the vagina. If the vaginal portion of the Müllerian ducts is differentiated from the outset by being solid and remaining solid longer than the other portion of the ducts, it remains an anatomical fact that the possibility of the vagina remaining solid is supported even with a normally developed uterus present. Should the opinion of Berry Hart prove correct, namely, that the vaginal epithelium does not arise at all from the Müllerian ducts, the entire argument of Nägel falls to the ground; for formed membranes which, according to Nagel, measure several millimeters in thickness and contain blood vessels, may be congenital. This is conceded by Nagel. Veit is of the conviction that hematosalpinx associated with gynatresia is due to an infection; otherwise, the necessary closure of the tube cannot be explained. The contents of a hematosalpinx, according to him, are the products of bacterial inflammation of the tube. That this is not essentially true, is proved by analogy with tubal pregnancy where, without infection, the escape of aseptic blood into the peritoneal cavity may cause adhesion of the fimbriated end of the tube. Veit has, moreover, acknowledged that atresia of the abdominal end of a tube may certainly occur without infectious processes.

With these intellectual giants at variance, how shall I classify my case? With nearly a total absence of the vagina and normally developed uterus and appendages, the Nagel-Veit theory would compel us to label it as an acquired vaginal atresia. On the other hand, the complete absence of the hymen, the distinctly undeveloped *labia minora*, as well as the smallest evidence of a vestibule, give us reason to suspect a lack of development in the lowest portion of the genital tract. Besides this, the argu-

ments of Krönig-Doderlein seem to me to have quite some force. In addition to this, the presence of peritonitis was clearly not associated with any possible recent infection. There were no intestinal adhesions which I recall to permit of the migration of bacteria directly from the intestine into the blood-sacs, and it is more than likely that, if a primary inflammation in the genital tract of an infectious character ever did exist, it must have been at so ancient a date that the infectious elements had died a natural death long before the onset of menstruation. Again, in the three cases of true congenital absence of the vagina with more or less complete absence of uterus and appendages, which have passed under my observation (two of which were published in the *Am. Journ. of Obstetrics;* the third still unpublished), there was an interlabial partition suggestive of even a primitive vaginal cul-de-sac which was not present in this case. And, lastly, with complete development of the uterus and adnexa, determined by laparotomy, and a distinct congenital malformation of the clitoris (case published in *Gynäkologische Rundschau*, January, 1908), there was a distinctly undeveloped hymen, vestibule and vaginal canal. A consideration of these facts makes me also doubt the correctness of the Nagel-Veit theory, and seems to justify the placing of our cases in the category of congenital atresiae.

From a practical point of view it does not make much difference whether the absent vagina is dependent on error in union of the Müllerian ducts or whether an embryonal or post-fetal vaginitis has determined the agglutination and effacement of the vaginal lumen. I have, therefore, in this study, dispensed with the term "*congenital*" atresia altogether and have designated, in its stead, "*primary*" atresia. The true "*acquired*" atresia, *i.e.*, that occurring after birth and attributable usually to a vaginitis of infectious, traumatic or other nature, I have qualified as "*secondary.*" Thus the gynatresiæ may be classified as follows:

I. *Primary Blood-retention*:
   (a) malformations of Müller's ducts;
   (b) adhesions acquired in embryonal or early infantile life.

II. *Secondary* Blood-retention:
   A. Non-puerperal.
      (a) traumatism (operations, neglected pessaries, injuries, etc.

(b) infections (scarlatina, small-pox, diphtheria, gonorrhea, etc.);

(c) natural (old age, atrophy, chronic catarrhal inflammations);

(d) mechanical (ball-valve tumors in the uterus, pressure of tumors at uterine cornua on tubes, uterine flexions, carcinomata, etc.);

B. Puerperal.

inflammations with ulceration or gangrenous destruction of cervical or vaginal mucosa.

The question has been often discussed as to the source of the blood in the Fallopian tube in those cases complicated with hematosalpinx. That there should be an accumulation of blood in the uterus (hematometra), in the cervix (hematotrachelos), and in the portion of vagina still patent (hematocolpos), is quite clear when it is borne in mind that the menstrual blood has no means of escape. But the blood accumulation in the tubes has not found a ready explanation. There can be no doubt that the reflux of blood from the uterine interior into the tubes is the underlying cause in probably all of the cases at the outset. Autopsies and examinations of specimens have shown, however, that after a while not only is the fimbriated end of the Fallopian tube shut off and occluded (as happens in hydrosalpinx or pyosalpinx, and by the same mechanism of an irritative peritonitis), but that all communication with the uterine cavity is similarly to a greater or lesser degree blocked. Hence, the "reflux" explanation does not elucidate all the cases or even the same case at different stages. It has been shown that, under certain conditions, blood may exude from the tubal mucosa itself. The theory of vicarious menstruation is very inviting, and is the one on which I explained the presence of two large ovarian blood tumors which I removed eight or ten years ago from a woman who had suddenly ceased to menstruate during four or six months, and who suffered the most intense cramps at each menstrual epoch.* This theory of vicarious menstruation is accepted by most authorities (Pozzi, Fritsch, Mann, Martin, Keating and Coe, Bandl, Rose, Ashton, and others). On the other hand, simple circulatory disturbances in the mucous membrane of the tube and resulting, perhaps, from pressure of the accumulated blood in the uterine interior

Med. Record, Aug. 19th, 1899.

(Breisky), may explain the constantly increasing collection of blood in the tubes. Stratz assumes a tubal atresia resulting from mechanical causes due to circulatory disturbances or infections. While not denying the possibility of a reflux of blood from the uterine interior, he places this in the second position. In the first place, he accounts for the blood accumulation in an hematosalpinx principally as due to the edema and swelling of the mucous membrane deprived of its epithelium in the occluded tube. According to Mann (also Bandl), the blood of a hematosalpinx, in a few instances, was apparently derived from a ruptured Graafian vesicle, the fimbriated extremity of the tube being fused with the ovary, as in cases of tubo-ovarian cyst. Bandl suggests that at times an apoplectic focus in the tube may rupture through the mucous membrane and produce a hematosalpinx. Frankly confessed, I believe that, while some of these explanations may clear up the origin of certain varieties of hematosalpinx, I hardly think that they apply to those associated with the gynatresiæ. I believe that uterine regurgitation and tubal menstruation will satisfactorily explain the presence of the hematosalpinx (as well as hematovarium which, I think, is always secondary to the hematosalpinx) in these cases.

I have just a word to say in regard to symptomatology. Up to the age of puberty there is ordinarily no complaint. Menstrual molimena—with abdominal cramps as the main symptom—will be the first evidence of the condition. During the menstrual intervals, the patient will again be in her normal condition of health. As time goes on, other symptoms will appear. The pain may now become continuous, because blood may trickle into the peritoneal cavity, causing a pelveo-peritonitis of greater or lesser extent. Pressure symptoms may arise and the patient may notice the formation of a tumor in the abdomen. As Hegar and Kaltenbach express it, once a retention blood tumor from an occlusion of the genital canal has formed, "the patient is always in danger of peritonitis or rupture of the sac."

The retention of blood as a result of hymeneal atresia is of common occurrence and occupies a very minor place in the present paper. Personally, I have had four such cases pass through my hands, as well as several atresiæ of the cervix,

which I have never thought worth the trouble to publish. So, I imagine that most gynecologists see quite a number of these cases, and I know that they are often enough handled successfully by the family doctor. So that while a certain number of them will be considered, it must be understood that they represent, in all probability, only a very small proportion of the cases which are actually met with. In this paper, in fact, I am mostly interested in blood-retention when due to atresia of the vagina and cervix.

In the year 1888, Stephen Fuld wrote a classical paper entitled " Salpingotomie wegen Hematosalpinx bei Gynatraesie " (*Arch. f. Gynäck.*, 1889, XXXIV, p. 19). After going thoroughly over the literature of the subject, he proved that the management of the blood retention tumors of the gynatresiæ associated with hematosalpinx, up to that time, was entirely wrong. He pointed out the fact that the fearful mortality-rate in these cases—with or without operation—was due to the rupture of an hematosalpinx. He, therefore, advised the deliberate performance of laparotomy and salpingotomy in order permanently to exclude the immediate and remote dangers arising from the presence of an hematosalpinx. Briefly stated, he collected 65 cases of gynatresia with hematosalpinx—some acquired but most of them congenital—and found the frightful mortality of 74 per cent. Of the 48 fatal cases, nine died without operation. In 27 of the fatal operations the hematosalpinx occurred in a single genital canal. In the remaining 12 cases the utero-vaginal canal was double. Although not strictly correct and open to criticism—particularly in regard to the single-horned uterus—Fuld divided his cases into two classes: those with a single and those with a double genital canal. The classification is practical, and, in conjunction with most writers on the subject, I shall adopt it.

Since this paper by Fuld was written—about 20 years ago—the literature on the subject of blood-retention due to the gynatresiæ has piled up enormously. The writer has attempted to go over this material and bring the subject up to date. As this work had to be accomplished within a fixed time-limit, and as some of the monographs and papers were inaccessible or published in languages beyond the writer's comprehension, he begs to apologize for the shortcomings of the present essay.

Without attempting to re-hash in detail the writings of Breisky, Neugebauer, Wagner, Rose, Puech and an army of indefatigable workers, I purpose mainly to take up the subject at the point where Fuld stopped and see what has been accomplished since his time.

From 1888 to the beginning of the present year, 1908, I have been able to collect 164 cases of blood-retention associated with gynatresia as follows:

I. Primary Blood Retentions:
(a) single genital canal.....80 cases;
(b) double genital canal.....65 cases;
II. Secondary Blood Retentions.....19 cases;

Total.....164 cases.

Spontaneous rupture or evacuation of primary blood-retention tumors with successful outcome has been known to take place externally through the obliterated area, or into the bladder or rectum, or through the septum of a double uterus. Fuld's paper refers to three such cases. My own researches have discovered seven more in primary blood-retentions and one in secondary blood retention [3, 10, 13, 29, 40, 44, 72, 164].* Most (possibly all) of these cases were not associated with hematosalpinx and presented an atresia of the hymen or lower end of the genital tube. During the past 20 years there have been no cases reported, to my knowledge, of spontaneous rupture of a hematosalpinx with fatal peritonitis.

Interventions for hematocolpos or hematometra in the single genital canal (leaving a possible hematosalpinx to chance.) According to Puech (Neugebauer), Felix Plater, on September 12, 1594, was the first to propose section of a vaginal atresia, but Roonhuysen was the first to do it. Dupuytren and Amussat, in the early part of the 19th century, established the operation of blunt dissection on a scientific basis, and this is the method still followed, with slight modifications, at the present day. Puech (*loc. cit.*) collected 66 cases of section of a vaginal atresia with four relapses. Of 28 cases of congenital atresia, there were two recurrences, two unfinished operations and of the 24 remaining cases, two deaths. In 300 cases of vaginal or cervical

* The numbers in brackets refer to the tabulated cases at the end of this paper.

atresia, hematosalpinx occurred 30 times. Fuld describes seven successful operations in the single genital canal, in which hematosalpinx was thought to be present. Neugebauer insists that Amussat's case be added. One operator, Rope, passed a catheter through the uterine cavity into the distended Fallopian tube and squeezed out the bloody contents—a method which Fuld properly condemns. Breisky released the blood successfully in two cases, complicated by hematosalpinx, through a broad vaginal incision past the obliterated area. Without hematosalpinx Robert Meyer (1896) collected 18 cases of broad vaginal atresia operated on with 17 recoveries; with hematosalpinx, 38 cases and 25 deaths. Since 1889 I find only four such cases of hematosalpinx which were cured (or disappeared) after cutting through the vaginal obstruction (2, 3, 10, 13) and one case (first of Horrock's cases [73] see remarks), which ended fatally. Of course, in this period of time, there is a large number of cases reported—and probably a great many more which were never reported—in which primary blood retentions in the single canal, without hematosalpinx, were successfully operated on from below (30 cases): [5, 6, 9, 11, 12, 15, 16, 21, 24, 26, 28, 35, 36, 41, 42, 43, 46, 48, 49, 50, 51, 58, 59, 60, 61, 62, 63, 75, 76, 79]. One case [45] later required a double salpingo-öophorectomy; in two cases [69 and 80] the blood-sac was opened through a laparotomy wound; and in one case [62] a hydrosalpinx was later punctured and emptied from below.

The double genital canal is often the seat of blood retentions, usually in the form of hematometra unilateralis with or without hematosalpinx. Thus, Fuld reports four cases which terminated favorably, after puncture per vaginam, and 12 cases which died. Wagner (1896) was able to collect 43 cases, of unilateral hematometra with the following record:

	Cases.	Died.	Recovered.
Not operated	6	5	1 (?)
Punctured or incised from below	26	10	16

Kehrer (1900) includes in his list of 12 cases of hematometra in a rudimentary accessory horn, one which was discovered at autopsy, one in which the blood-sac was emptied by compression and two which were emptied by puncture per vaginam. The others were treated by laparotomy and will be referred to later. In five of these 12 cases a hematosalpinx was said to

be present. Of course, these statistics are to a great extent repetitions of the same cases quoted by other writers and hence apt to be misleading. The most recent collection of cases of retention of menstrual blood in the double genital canal is that of Gross and consists of 90 cases, which are tabulated as follows:

57 cases of lateral hematometra;
5 " " " " with partial superior hematocolpos;
24 " " " hematometra with total hematocolpos.
4 " " " hematocolpos without hematometra;

The cases successfully operated on from below, between 1889 and 1908, according to my own researches, are as follows:

Double uterus with atresia vaginæ, six cases [93, 97, 111, 117, 140, 145];

Double uterus with atresia cervicis, three cases [83, 89, 101].

In three of these cases an exploratory laparotomy was first done before the diagnosis was absolutely established [83, 97, 145]. In the same space of time I find reports of two fatal interventions, the histories of which I abstract in as short a space as possible.

Drake Brockman [82] describes a case of retention of the menses, during a period of seven years, in a girl with imperforate hymen and septate uterus. An incision in the obstructing membrane was followed by oozing of blood during a half hour. The patient died 26 days later. No autopsy.

Dalmon and Monnet [130] describe a case occurring in the service of Chaput. A girl with hematocolpos, imperforate hymen and atresia of the lower vaginal segment, was operated in the usual manner by a vaginal dissection which had to be made to a depth of five or six cm. before the blood-collection could be reached. After evacuation, the upper vagina was sutured to the labia below. On the third day fever with thready pulse. A mass felt in the rectum was now punctured. Death a few hours later. The autopsy showed that a right-sided hematosalpinx had been opened by the previous operation. There were no signs of peritonitis and the pedicle of the tube was twisted. Chaput attributes death in this case (as well as to another similar experience six years previously) to " re-

flex intestinal occlusion " brought on by torsion of the tube. In both this case and the earlier one [38] Chaput thinks the torsion of the tubes was consequent to emptying of the blood-tumor and fixation of the distal end of the tube; the torsion occurring by a revolving motion, such as is produced in a handkerchief when turned in the same direction between the hands.

It is clear, by this time, that the great danger of the blood-retentions lies in the regions beyond the uterine interior, in the single uterus, and in a shut-off cavity in the double uterus. To Schroeder, (1881), belongs the glory of having been the first deliberately to perform a laparotomy in such a case.

The patient was a girl, aged 17, who had been menstruating for three years. Since 18 months she had suffered abdominal pain. The following diagnosis was made: Hematometra of the left horn of the uterus, left heamatoslpinx, and uterus bicornis partum septis. At the time of the operation Schroeder intended to remove the left half of the uterus but, as there seemed to be a rupture of the hematometric blood into the connective tissue, he contented himself with removing only the blood-filled tube. Later, there was a spontaneous discharge of chocolate-colored blood through the vagina. This opening closed prematurely and required subsequent incision and drainage.

The three cases of successful laparotomy reported by Fuld, in 1889, for hematosalpinx in the single genital canal are criticized by Pozzi as being inconsistent, because in two of them (cases of Terillon) the atresia was not congenital but acquired. In the double genital canal with hematosalpinx, Fuld was able to report two successful laparotomies in addition to the third one done by Kehrer (which gave the stimulus to write his paper). Leopold adds two previously unpublished cases in which laparotomy was done respectively once and twice in two patients, one patient having hematosalpinx and the second patient having an occluded right horn filled with blood. In the second case the blood-filled horn was left to its fate, both adnexa only being removed. The two patients recovered. The moderate hematometra in the first case and the pent-up blood in the uterine horn in the second case underwent spontaneous absorption. In Wagner's 43 cases, previously alluded to, of hematometra unilateralis, celiotomy was done in 11—all of the

cases making a good recovery. In 1898, v. Rosenthal was able to add seven more successful laparotomies in cases of this kind. Saenger, in 1896, collected 11 cases (according to v. Rosenthal) of hematosalpinx complicating this condition for which adnexal ablation was successfully done. v. Rosenthal adds six more, in several of which the offending horn was simultaneously removed.

My own series (collected since 1889) of *laparotomies* (excluding hysterectomies) for *hematosalpinx in the single genital canal with primary atresia* of the hymen or vagina, is as follows:

Successful operations:
- Incision of hematosalpinx evacuation of bloody contents, with immediate suture [68]..... 1 case
- Salpingo-öophorectomy and salpingostomy [71]..... 1 case
- Salpingotomy or salpingo-öophorectomy [1, 7, 20, 27, 37, 39, 47, 52, 53, 55, 56, 73].....12 cases
- Incision of hematometra, evacuation of bloody contents. Salpingotomy or salpingo-öophorectomy [67]..... 1 case

Total.....15 cases

Unsuccessful (fatal) operations:
- Salpingotomy or salpingo-öophorectomy [19, 23, 38, 40], total..... 4 cases

So that, in this classification, we find four deaths in 19 cases operated on during the past 20 years.

The four fatal interventions for hematosalpinx in the single genital canal with primary atresia are herewith briefly given:

C. C. Webster (1895), young girl, from Iceland, who had never menstruated, with history of retention covering three years. Atresia vaginæ of the lower one-quarter. Hemato (pyo) colpos; hemato (pyo) trachelos; hematometra (?); left hematosalpinx. Laparotomy, Peritonitis present. Distended left blood-filled tube opened and emptied. Double salpingo-öophorectomy. Death a few weeks later from septic peritonitis. The pelvis and thorax were secured and frozen. Vertical mesial sections were made, from one of which a beautiful illustration is printed. The *uterus was enlarged to* $4\frac{3}{8}$ *inches.* Its walls were very much thickened and its cavity was empty. *The cervix and upper occluded vagina were distended with "thick, greenish fluid and fibrinous septa."*

C. Martin (1896). Atresia vaginæ (in upper $\frac{3}{4}$) and cervix.

Hematometra. Double hemato (pyo) salpinx. Vagina artificially dissected up to the uterus. The blood-collection in the uterus was later incised, evacuating over a quart of thick, black blood. Suture of uterus to vaginal mucous membrane. *Septic peritonitis* at the end of a week. Laparotomy. *Tubes distended with green, fetid pus* and adherent to the intestines. Double salpingo-öophorectomy. Death 42 hours later. Martin adds that the tubes originally contained blood, which became infected through adhesions to the intestine; but it seems to me more likely that the infection was post-operative and came through the newly-opened vaginal tract.

Chaput (reported by Morely, 1898). Imperforate hymen; retro-hymeneal vaginal septum. Hematocolpos. Puncture of membraneous diaphragm evacuating ½ litre of blood. Laparotomy 48 hours later for collapse symptoms of peritoneal origin. *Abdomen contained* 200 *grammes of black blood.* Double salpingotomy. *The right tube was the size of a fist and twisted on itself in a special manner six times, the left tube showed five spiral twists.* The twisting of the tube was attributed to the effects of removing the blood-supporting column from below. The patient died in a short time after the operation.

Albertin (1899) reports a case of atresia vaginæ with hematometra. Three years previously the rudimentary vagina was punctured without benefit. Vaginal dissection was attempted when the peritoneal cavity was unexpectedly invaded. The uterus seemed to be detached from the vagina. The wound was now packed in order to agglutinate the uterine fundus to the vaginal opening. Six days later the hematometra was punctured, giving exit to 50 or 60 grammes of prune-juice-colored blood. This was followed by exsection of a portion of the uterine wall to secure a permanent opening, and the wound was packed with gauze. The passage-way closed up later with pus formation, and a spontaneous opening in the rectum ensued. The patient, after this, menstruated per rectum for 18 months, when laparotomy was done. Tubo-ovarian abscesses were found and removed. Peritonitis set in on the fourth day, and the patient succumbed on the sixth day.

In reviewing these four fatal cases, one feels that the three last would have stood a better chance for recovery had laparotomy been done at the outset, before the deeper portions

of the genital tract had become infected. The results might have been entirely different, and three recoveries instead of three deaths might have been recorded. In other words, it is hardly fair to attribute these deaths to an operation which might have been done deliberately and in "clean" surroundings with everything to favor a successful outcome. Laparotomy as a "*dernier ressort*" is an entirely different operation from laparotomy done with deliberation and before infection has had a chance to appear. The case of Webster similarly accentuates the fact that it is *not always safe to rely on Nature for the absorption of the blood of a hematometra or hematocolpos*, and that a hysterectomy under similar circumstances—possibly with drainage at the lower portion of the abdominal wound—might have given this girl a better chance than she had. Of course, it is easier for an outsider to offer suggestions and weigh probabilities after an operation has been done; but as this paper is written in a purely scientific spirit, no offense is intended in criticizing the results of men whose reputations for thorough, clean and able work have long since been established and sustained.

In secondary atresia of the single genital canal without hematosalpinx, blood collections have, according to my studies in the period of time which my paper covers, been *successfully attacked from below* 10 *times*: [146, 148, 150, 151, 155, 157, 159, 162, 163, 164]. There were no deaths. Even a case of Hirst [152], in which a blood collection was unsuccessfully punctured per rectum, recovered after a subsequent hysterectomy. In this *secondary group* of cases (the really *acquired* type) I note *five cases with hematosalpinx* [149, 152, 156, 158, 161] *which recovered after laparotomy.*

No cases of hysterectomy for blood retention in the single genital canal are mentioned by Fuld. Landau and Pick, only six or seven years ago, wrote: "The abdominal radical operation in gynatresia with single canal has hardly ever been done. Even in the double genital canal with gynatresia and hematometra, we find up to 1889 no abdominal radical operation and only six cases in which supra-vaginal amputation (removing the double uterus and adnexa) had been done for hematometra and hematosalpinx, the result of genital closure." *The credit for the first successful hysterectomy in the blood-retentions*

belongs to an American—John Homans—who (in the *Boston Med. and Surgical Journal*, Sept. 8, 1883) reported a supravaginal amputation of a uterus bicornis in a girl 18 years old. In 1901, L. Landau did the first *total* abdominal hysterectomy for blood-retention in the single canal. Since 1889 I find the following:

For *the blood-retention in the single genital canal due to primary atresia, abdominal hysterectomy has been done* 10 *times.* The following recovered:

Supravaginal hysterectomy [25, 34, 65, 70]	4 cases
Total hysterectomy [54, 57, 77]	3 cases
Total	7 cases
To this should be added my case (total hysterectomy)	1 case
Total	8 cases

With fatal result 3 *abdominal hysterectomies were done:*

Supra-vaginal hysterectomy [14, 17, 66]	3 cases

In reviewing the fatal cases we find that the autopsy in Rossa's case [14] showed a right sided congenital hydronephrosis with valvular endocarditis, in addition to the pelvic conditions. In Murphy's case, after preliminary vaginal puncture, hysterectomy was done. The " broad ligament was sutured to the abdominal wound " and hemorrhage was controlled by gauze packing. *Four months later the abdomen reopened was found to contain nearly four pints* (60 ounces) *of blood.* Abdomen was closed without doing anything. *Five weeks later the wound burst open again* with the discharge of much serous brown fluid. The patient refused (!) further intervention and died shortly afterwards.

In Moullin's case [66], the blood-retention cyst, after vaginal dissection, was punctured. The opening had closed three months later. Laparotomy and hysterectomy now done. Foul-smelling pus gushed from the cervical canal. Vaginal drainage. Death three weeks later. We agree with Moullin when he says: " There is no reason why, had hysterectomy been done in the first instance, the patient should not have made a good recovery."

For the blood-retentions in the single genital canal due to secondary atresia, hysterectomy has been done successfully five times:

Abdominal hysterectomy [152, 158, 160].....3 cases
Vaginal hysterectomy [153, 161].....2 cases

Total.....5 cases

I may, perhaps, be criticized adversely for including the case of Schwartze-Halle, reported by Chrysopathes [161]. In this case occlusion of the tubes, with resulting double hematosalpinx, was due to two uterine adeno-myomatous tumors which, in their development, had caused an occlusion of the uterine ends of both tubes. This case is unique in medical literature, and is an irrefutable instance of blood being directly exuded from the tubal mucosa, instead of being due to a regurgitation from the uterine cavity.

The operations in the double utero-genital canal with unilateral blood accumulations are summed up by Kleinhaus (1897) as follows:

1. Simple evacuation of the hematometra through the vagina.
2. Evacuation through the vagina with preceding or subsequent celiotomy, and removal of hematosalpinx (and ovary).
3. Celiotomy. Incision of hematometra and resection or exsection of its wall with suture (Werth).
4. Removal of tubal sac with a portion of the distended half of the uterus and suture of the sac to the abdominal wall.
5. Removal of appendages from both sides.
6. Supra-vaginal amputation of the uterus.
7. (We can add: total hysterectomy.)

Fritsch, Kerzmarsky and Kehrer sewed the blood-sacs into the abdominal wound in order to prevent the future escape of blood into the peritoneal cavity.

It is interesting to note that in the 90 cases of blood retention in the double genital canal, collected by Gross, there were four cases of lateral hematocolpos. Such cases are, of course, accessible to a simple vaginal incision. In the vast majority of the reported cases, however, the blood-sac consisted usually of the blood-filled half of a double uterus with or without hematosalpinx. E. Simon, in 1888, reported 22 cases complicated with hematosalpinx of which 14 died and eight recovered:

After vaginal incision,	5	recoveries,	11	deaths.
" rectal puncture,	1	"	1	"
" laparotomy,	1	"	0	"
Without treatment,	1	"	2	"

Up to 1898 v. Rosenthal was able to collect 18 celiotomies, done in cases of bicornate uterus, with 18 recoveries.

My own studies, covering the past 20 years, show the following results concerning *amputation of a blood-filled horn in the double uterus*:

Successfully operated by vaginal celiotomy [94]	1 case
Successfully operated by vaginal celiotomy [with hematosalpinx 114]	1 case
Successfully operated by laparotomy [85, 92, 104, 109, 112, 125, 127, 128, 129, 131, 132, 134, 141]	13 cases
Successfully operated by laparotomy (with hematosalpinx) [86, 91, 95, 96, 100, 110, 121, 136]	8 cases
Total	23 cases
Died after laparotomy (with hematosalpinx) [116, 137]	2 cases

There were, therefore, 23 *successful operations and two deaths*.

Hysterectomy (supra-vaginal or total) for unilateral hematometra was done 15 *times without a single death*:

Per vaginam [132]	1 case
Abdominal section [106, 113, 123, 126, 138, 142, 144]	7 cases
Abdominal section (with hematosalpinx) [102, 115, 118, 119, 135, 139, 143]	7 cases
Total	15 cases

Including my own case, we find, therefore, in the past 20 years, 23 cornual amputations and 28 hysterectomies done on the double uterus—a total of 51 operations with five deaths. Of 16 cases complicated with hematosalpinx, there were two deaths. How this compares with the figures of Fuld or of E. Simon, who 20 years ago, in 18 cases treated by the older methods, found 12 deaths, will readily be appreciated. Nothing could more positively prove the correctness of Fuld's position at that time when he positively stated that laparotomy must be the treatment of the future.

While Fuld limited himself to advising the removal of the blood-sac in hematosalpinx and thus, at one stride, wiped out the fearful mortality which prevailed down to his time, the question arises to-day whether we are to limit ourselves to a salpingotomy (or exsection of the blood-filled horn in the double uterus); whether we should go further and do a hysterectomy, or whether we should content ourselves with splitting

open the blood-filled sacs, and, after wiping out their contents, seek to make proper provision for the escape of menstrual blood through a new vaginal canal. Theoretically, the last plan is the one to elect, as it implies the retention of the sexual organs and the continuation of their function. Experience, however, has taught this to be seldom practicable, in spite of the excellent work of Zweiffel, Hofmeier, Tuffier and others. The removal of the tubes and ovaries, leaving the blood-filled uterus (and rudimentary vagina), I believe to be less desirable than the radical operation. Where, however, from technical difficulties it may not be possible to remove the uterus with the blood sacs, it will always be justifiable either to remove the adnexa alone or, in addition, to attach the uterine sac to the abdomen, or to the vagina when a patent and permanent canal can be properly made.

In regard to the hematovarium, which constituted such an enormous tumor in my case, I have looked over the 164 cases which form the basis of this paper and find that it constituted part of the retention sac in 16 cases. In five cases it was situated on the right side. In six cases it was situated on the left side. In four cases it was found on both sides. In one case its situation was not stated.

PRIMARY BLOOD-RETENTION IN SINGLE CANAL.

No.	Reporter or Operator.	Reference.	Diagnosis.	Treatment.	Result.	Remarks.
1.	G. Leopold (1885) (Case I)	*Arch. f. Gyn.* 1889, p. 371.	Atresia vaginæ; moderate degree of hematometra; temporary right hemato-salpinx; permanent left hematosalpinx.	Blunt dissection of vagina to depth of 3 cm. when operation abandoned. Laparotomy four months later. Uterus and right ovary enlarged. Right tube moderately thickened. Salpingo-öophorectomy. Left tube distended and punctured (contained much blood); encircled a cystic ovary. Salpingo-öophorectomy.	Recovery.	The left hemato-salpinx was ready to burst. Uterus was not disturbed and the contained blood was evidently absorbed.
2.	*Ibid.* (1888) (Case III).	do.	Hymen bipartitus; atresia vaginalis inferioris partis; hematometra; hematosalpinx duplex.	Incision vaginal cul-de-sac (1 cm. deep) to globular tense swelling; silk sutures to edges; puncture of blood accumulation (1½ litres tarry liquid). Glass tube for drainage. Two weeks later blunt dilation of new vaginal tract. Glass tube retained.	Recovery.	Four weeks later no evidence of distended tubes.
3.	A. Turreta.	*Gior. interna z d. Sc. med. Napoli.* 1889 U. S. xi, p. 21.	Hematometra due to deeply-seated vaginal atresia. Hematosalpinx?	Vaginal incision.	Recovery.	Several pelvic tumors spontaneously disappeared; one remaining sac opened into the bladder.
4.	Cospedal Tonie.	*Index Medicus* (1889)	Hemato colpos and hematometra.			
5.	Mongonidi.	*Ibid.*	Partial congenital atresia vaginæ; hemato colpometra.	Operation (vaginal incision).	Recovery.	
6.	Rosinski, Jr.	*Allg. Med. Cent, Ztf.*, 1892 LXI, 2042.	Hymen imperforatus; hemato-colpos.	Probatory puncture; incision.	Recovery.	
7.	A. F. Currier.	*N. Y. Jour. of Gyn. and Obst.* 1893, iii, p. 1087.	Absence of vagina (con genital); hematometra	A year previously puncture per rectum. Subsequently a false passage made at vagina. Again retention of menses. Laparotomy with removal of congested and hypertrophied adnexa. Another vaginal opening with evacuation of uterine contents. During subsequent efforts at dilatation bladder injured; spontaneous closure of vesico-vaginal fistula.	Recovery.	

PRIMARY BLOOD-RETENTION IN SINGLE CANAL.—*Continued.*

No.	Reporter or Operator.	Reference.	Diagnosis.	Treatment.	Result.	Remarks.
8.	Hochenburger.	*Ztsch. f. Gebtsh.u. Gyn.* 1893, XXVI, p. 60.	Atresia vaginalis (hymenalis?); hematocolpos.	Exsection of hymen; suture of its edges.	Recovery.	No changes in uterus, tubes or ovaries.
9.	*Ibid.*	*Ibid.*	Hymen imperforatus; hematocolpos.	Incision of hymen.	Recovery.	Epileptic convulsive seizure before menses appeared.
10.	*Ibid.*	*Ibid.*	Hematocolpos; double hematosalpinx; atresia hymenalis.	Resection of hymen; blood-accumulation discharged in several sittings; tubal swellings still evident after 14 days; nothing further done.	Recovery.	After 6 weeks right tubal swelling disappeared and left felt considerably reduced in size; one year later nothing found; 3½ years later patient married and had one normal delivery.
11.	F. C. Barker.	*Indian Med. Chir. Review.* 1893, I, p. 140.	Atresia vaginalis; hematocolpos; hematometra?	Incision per vaginam.	Recovery.	Later lost from observation.
12.	J. C. Da Costa.	*Med. News,* 1894, p. 269.	Absence of vulva and vagina; hematometra.	Perineal incision followed by trocar and dilator. Glass plugs worn.	Recovery.	From urethra to anus a firm fleshy mass without any sign of hymen or vagina. No tendency of artificial opening to close a year later.
13.	Ettore de Minicis.	*Gaz. degli ospedal e delle clin.,* 1894, XV, p. 339.	Atresia hymenalis; hematocolpos; hematosalpinx.	Incision of bulging imperforate hymen. On fourth day an elongated tumor to left of uterus felt per rectum size of pigeon's egg (was this not rather an enlarged ovary instead of a tube?) The hematosalpinx (?) emptied itself spontaneously through ostium uterinum.	Recovery.	
14.	Emil Rossa.	*Zent. f. Gyn.,* 1894, xviii, p. 422.	Vaginal atresia; cyst ovarium bilateral hemorrhagicum; left hematosalpinx (retrograde).	Laparotomy. Bladder injured. Uterus not much enlarged but intimately adherent to two ovarian cystic blood tumors. After puncture removal of tumors with uterus and drainage through Douglas' sac.	Death after 48 hours.	Post mortem. Hydronephrosis congenital dextra. Congenital atresia of upper portion of vagina and cervix. Valvular endocarditis. Peritonitis and pericystitis.

PRIMARY BLOOD-RETENTION IN SINGLE CANAL.—*Continued.*

No.	Reporter or Operator.	Reference.	Diagnosis.	Treatment.	Result.	Remarks.
15.	Lengor.	*Ann. de la Soc Belge di chir.*, 1895, iii, p. 396.	Atresia vaginæ; hematometra.	At 9 cm. from the solid membrane occluding the vulvar orifice the cervix was reached after blunt dissection between rectum and bladder. Emptied hematometra reaching above umbilicus.	Recovery.	Condition good several months later.
16.	Don F. de L. Recio.	*Anal. de Obst. Ginecop. y Pediat.*, 1895, CLXX vj., p. 201.	Complete vaginal atresia; hematometra.	Vertical incision between labia 3½ cm. long. Deep dissection with grooved director through an obstruction of 5 or 6 cm. in thickness.	Recovery.	Very difficult operation. No report after first menses.
17.	J. Murphy.	*Br. Med. J.*, 1895, I, p. 65.	Imperforate hymen (?) (Probably absent vagina.) Imperforate os uteri; hematometra.	Laparotomy. Puncture of uterus (10 oz. brown serum and large organized blood-clot in its interior). Hysterectomy. Broad ligament sutured to abdominal wound. Gauze packing for hemorrhage. Four months later abdomen reopened and full of organized blood-clot (60 oz.). Abdomen closed. Five weeks later wound burst open with discharge of brown serous fluid. Patient succumbed shortly afterwards.	Died.	Operative technique is open to serious criticism and may have had something to do with the unfortunate result.
18.	Barone.	*Index Medic.*, 1895.	Hematometra and hematosalpinx, etc.			
19.	C. Webster.	*Am. J. Obst.*, 1895, XXXII 544.	Atresia vaginæ (lower ¼); hemato (pyo) colpos; hemato (pyo) tracheolos; hematosalpinx sinistera; hematometra (?)	Laparotomy. Peritonitis present. Distended left tube (hematosalpinx) first opened; then double salpingo-oophorectomy.	Death several weeks later from septic peritonitis.	Vertical mesial section from frozen pelvis showed uterus enlarged to 4⅜ inches; walls very much thickened; cavity empty. Cervix and upper vagina distended with "thick, greenish fluid and fibrinous septa."
20.	do. (Simpson).	*Ibid.*	Complete atresia vaginæ; hematometra; hematosalpinx adherent to posterior uterine surface.	Artificial vagina made; uterine cavity opened and blood evacuated. A year later vagina contracted and cervix closed. Aspiration through cervix of uterine contents. Several months later laparotomy with partial excision of cystic mass behind uterus.	Recovery.	

PRIMARY BLOOD-RETENTION IN SINGLE CANAL.— *Continued.*

No.	Reporter or Operator.	Reference.	Diagnosis.	Treatment.	Result.	Remarks.
21.	Wm. H. Meyers.	*Trans. Am. As. Obst. & Gyn.*, 1896, IX, p. 302.	Complete absence of vagina; hematometra.	Blunt vaginal dissection to depth of index finger; uterus punctured with trocar and opening enlarged with blunt pointed bistoury.	Recovery.	Temperature ranged between 102° and 103° (F) during four days; horseshoe pessary to keep wound open.
22.	Matvieyeff	*Index Med.*, 1896.	Congenital closure of vagina by hymen; hematocolpos.			
23.	C. Martin.	*Br. Med. Jour.*, 1896, II, p. 1289.	Atresia vaginæ (upper ¾) and cervix. Hematometra. Double hemato (pyo) salpinx.	Vagina dissected up to uterus. Latter incised, evacuating over a quart of thick blood. Suture of uterus to vaginal mucous membrane. Septic peritonitis a week later. Laparotomy. Tubes distended with green fetid pus and adherent to intestines. Adnexa of both sides removed. Patient succumbed 42 hours later.	Death.	Martin thinks the tubes originally contained blood which became infected through adhesions to the intestines.
24.	E. K. Kellenberger.	*Kansas City Med. Ind.*, 1896, xvii, p. 20.	Atresia vaginæ (lower portion); hematocolpos hematometra?	Aspiration of 30 ounces of blood, leaving canula in situ. Ten days later opening enlarged on a director followed by the use of a Goodell dilator. Then free incision.	Recovery.	
25.	C. Martin.	*Med. Press & Circular*, 1896, ii, p. 571.	Atresia vaginæ (lower ¾); hematocolpos (upper ¼); hematometra; double hematosalpinx.	Laparotomy. Hematometra (size of six-months' pregnancy) evacuated by median incision and wiped dry. Hystero-salpingo-oöphorectomy (amputation of uterus at level of internal os). Cervix sutured at lower angle of abdominal wound. Glass drain. Mucus fistula left.	Recovery.	
26.	Amand Routh.	*Br. Med. Jour.*, 1896, II, p. 1131.	Atresia vaginæ; hematometra.	Artificial vagina constructed. Unsatisfactory result because of great difficulty in keeping vagina patent.	Recovery.	Considered castration as proper treatment in these cases of persistent menstruation, without proper menstrual exit.

PRIMARY BLOOD-RETENTION IN SINGLE CANAL.—*Continued.*

No.	Reporter or Operator.	Reference.	Diagnosis.	Treatment.	Result.	Remarks.
27.	Decio.	*Rasseg. d'ostet. e ginec.*, 1896, V, p. 398.	Complete (?) absence of vagina; hematometra and double hematosalpinx.	Failure of attempt to reach cervix through artificial vaginal route. Laparotomy. Double hematosalpinx removed after emptying the sacs. Gauze drainage.	Recovery.	
28.	E. H. Lee.	*Medicine*, 1896, ii, p. 534.	Vaginal atresia (low) and hematocolpos.	Blood-collection evacuated after puncture. Later, opening enlarged, perineum split and vaginal mucosa stitched to skin.	Recovery.	
29.	Vitrac.	*Rev. Med. Chir. des Maladies des femmes*, 1896, xviii, p. 485.	Complete absence of vagina; hematometra; hematosalpinx.	Vaginal cul-de-sac 1 cm. deep. Perforated hymen (?) allowing pent-up blood to escape.	Recovery.	Diagnosis of hematosalpinx made from a digital exploration per rectum and supposed to have spontaneously disappeared.
30.	Novitski.	*Index Medic.*, 1897.	Hematometra.	Laparotomy.		
31.	Zaks, G. G.	*Index Medicus*, 1897.	Atresia vaginalis partialis; hemelytrometra.	Operatio Gynatresial.		
32.	L. Varnek.	*Ibid.*	Atresia vaginæ; hematometra; double hematosalpinx.			
33.	Plieshkoff.	*Ibid.*	Congenital atresia vaginæ; hematocolpos; hematometra and double hematosalpinx.			
34.	W. W. Beckett.	*So. Califor. Pract.*, 1897, xii, p. 447.	Atresia cervicis (congen?); hematocolpos; hematometra; left hematosalpinx; double ovarian cysts (blood?).	Uterus amputated at level of internal os; removal of left hematosalpinx and ovarian cyst; also of tube (?) and ovarian cyst on right. Through vagina hematocolpos emptied through incision into cervix followed by gauze drainage.	Recovery.	
35.	J. F. Lynch.	*Virg. Med.*, semi-monthly 1897-8, ii, p. 7	Atresia vaginæ (middle ⅓); hematocolpos and hematometra.	After dissection through vaginal obstruction for a distance of one inch blood accumulation evacuated.	Recovery.	

PRIMARY BLOOD-RETENTION IN SINGLE CANAL.—*Continued.*

No.	Reporter or Operator.	Reference.	Diagnosis.	Treatment.	Result.	Remarks.
36.	F. Huc.	*La Normandie Medic.*, 1897, xii, p. 198.	Atresia vaginæ (septum); superior hematocolpos; hematometra.	Blunt vaginal dissection for 3 or 4 cm.; evacuation of 1 litre of blood after puncture with trocar vaginal mucosa sutured to vulva.	Recovery.	
37.	A. H. Ferguson.	*Am. Jour. of Surg. & Gyn.*, 1898, XI, p. 23.	Atresia cervicis; hematometra; double hematosalpinx.	Two adherent cysts discovered but not removed in a laparotomy done 5 years previously. Second laparotomy. Double salpingo-öophorectomy. Cervix split transversely per vaginam. Drainage of uterine cavity.	Recovery.	The probe introduced through opening in uterine horn passed beyond the internal os. Patient well one year later.
38.	M. P. Morely (Chaput).	*Bul. de la Soc. Anat. de Paris*, 1898, xii, p. 619.	Imperforate hymen; retro-hymenal vaginal septum; double hematosalpinx with twisted pedicles.	Puncture of hymen evacuating ½ litre of blood; laparotomy 48 hours later; abdomen contained 200 grammes black blood. Double salpingotomy.	Death.	Right tube was size of fist and spirally twisted on itself six times; left tube shows 5 spiral turns. The spiral turns are attributed to the effects of the removal of blood from below.
39.	Mainzer (L. Landau)	*Arch. f. Gyn.*, 1898, lvii, p. 681.	Atresia hymenalis; hematocolpos; hematometra; hematosalpinx.	Laparotomy. After inspection of conditions present hymen is punctured giving exit to ¾ litres blood. Right salpingotomy.	Recovery.	
40.	Albertin.	*La Prov. Med.* 1899, xiii, p. 75.	Atresia vaginæ; hematometra.	Three years previously rudimentary vagina punctured without benefit. Vaginal section. Unexpected entrance made into peritoneal cavity. Uterus felt detached from vagina (?); packing; puncture of uterus six days later evacuating 50 to 60 g. prune-juice colored blood; exsection of portion of uterine wall and packing. Later the wound closed with pus formation and recto-vaginal fistula (through which patient menstruated per rectum); 18 months later laparotomy for tubo-ovarian abscesses. Peritonitis on 4th day. Death on 6th day.	Death.	Thinks uterus was entirely independent of the vagina.
41.	R. Lofqvist.	*Mitth. aus d. Gynack. Klinik* (Engström, 1899-1901) iii, p. 261.	Congenital absence of vagina; hematocolpos; hematometra.	Vaginal dissection; evacuation of 1¾ litres blood.	Recovery.	

PRIMARY BLOOD-RETENTION IN SINGLE CANAL.—*Continued.*

No.	Reporter or Operator.	Reference.	Diagnosis.	Treatment.	Result.	Remarks.
42.	*Ibid.*	*Mitth. aus. d. Gynack Klinik* (Engström, 1899-1901, iii, p. 261.	Congenital atresia of lowest portion of vagina; hematocolpos.	Vaginal dissection to depth of 1½ cm. Evacuation of large quantity of blood.	Recovery.	
43.	*Ibid.*	do.	Congenital atresia of lower portion of vagina; hematocolpos; hematometra.	Vaginal dissection to blood tumor with discharge of tarry liquid.	Recovery.	Febrile convalescence due to retention.
44.	Broca.	*La semaine Gynec.*, 1900, V, p. 46.	Retro hymeneal vaginal obliteration; hematocolpos.	Spontaneous rupture in the night with escape of 3 litres of blood.	Recovery.	
45.	*Ibid.*	do.	Vaginal atresia (low); hematocolpos; hematometra (?)	Vaginal partition 1 cm. thick cut through allowing 1 litre blood to escape. Closure of opening. Fresh accumulation of blood. Laparotomy. Double salpingo-oophorectomy. *No blood in tubes.*	Recovery.	
46.	Jos. Godart.	*Bull. Soc. Belge de Gyn. et d'obst.*, 1900-01, xi, p. 39.	Cervical atresia (congenital); pyometra subesquent to hematometra.	The obstruction was torn through with the finger.	Recovery.	Post operative mental disturbances.
47.	Psaltoff.	*Ann. de Gynec et d'obst.*, 1900 LIV, p. 460.	Complete absence of vagina; right hematosalpinx.	Failure of vaginal operation. Laparotomy. Aspiration of blood tumor followed by removal of the sac. Left cystic ovary removed. Six months after operation menstruating per rectum began. Three years later the rectal bleedings recurred once every 3 months.	Recovery.	Believes that besides removing the ovaries the uterus should be regularly removed at the same time.
48.	T. J. Cullingworth.	*Br. Med. Jour.*, 1900, I, 65.	Atresia vaginæ; hematocolpos (low).	Vaginal dissection; 5 oz. brown liquid evacuated.	Recovery.	
49.	*Ibid.*	do.	Atresia vaginæ; hematocolpos (low).	Vaginal dissection; dilatation of opening; discharge thick brown liquid.	Recovery.	
50.	J. M. Fisher.	*Am. Gyn. & Obst. Journal*, 1900, xvii, p. 38.	Retro hymeneal atresia; hematocolpos; hematometra.	Vaginal incision evacuating 3 pints of tarry liquid; required subsequent incision with suture.	Recovery.	

PRIMARY BLOOD-RETENTION IN SINGLE CANAL.—*Continued.*

No.	Reporter or Operator.	Reference.	Diagnosis.	Treatment.	Result.	Remarks.
51.	F. W. Higgins.	*Med. News*, 1900, LXXVI, p. 200.	Atresia vaginæ; hematocolpos; hematometra.	Inicsion alone per vaginam?	Recovery.	
52.	O. Thienhaus.	*Jour. Am. M. Ass.*, 1901, xxxvii, p. 194.	Atresia hymenalis hematocolpos; hematosalpinx duplex; left hematovarium.	Laparotomy. Both blood-filled tubes and left ovarian blood-tumor removed. Small opening made into introitus vagina evacuating 2½ quarts of bloody liquid. Ten days later a plastic operation done to keep vagina open.	Recovery.	At time abdomen was opened blood was seen oozing from the abdominal end of the left tubal tumor.
53.	Sänger (Labusquiere).	*An. de Gynec. et d'obst.*, 1901 LVI, p. 141.	Absent vagina (congenital); hemato-trachelos; hematometra; double hematosalpinx.	Unsuccessful attempt to reach blood accumulation from below. Two months later laparotomy. Free blood in peritoneal cavity. Another attempt to reach uterus from below failed. Double salpingo-öophorectomy.	Recovery.	
54.	L. Landau & L. Fick.	*Arch. f. Gyn.*, 1901, LXIV, p. 98.	Atresia fornicis vaginæ; atresia of cervix "*ex hyperplasia mesonephrica*;" hematometra corporis uteri hyperplastic myomotosi; hematosalpinx duplex; salpingitis productiva duplex; hyperplasia ovarii dextri; kystoma hemorrhagicum ovarii sinistri.	Laparotomy. Right salpingo-öophorectomy Left hematovarium ruptured. Left salpingo öophorectomy. Unsuccessful attempt to open into vagina through Douglas' cul-de-sac. Uterus bisected and each half removed separately, opening a circum cervical mesonephritic adeno-myoma (containing blood). Vaginal drainage Peritoneum closed below and above.	Recovery.	Femoral phlebitis during convalescence. This woman had been previously (1883 or 1884) operated by Schroeder. Before this, at 18, an operation had been done, followed up by dilatation with sponge tents.
55.	Georg. Fleck.	*Mon. f. Geb. u. Gyn.*, 1901, xiii, p. 419.	Congenital absence of vagina; hematometra; double hematosalpinx.	Laparotomy. No trace of left ovary. Right ovary removed. Extirpation of blood tumors. Discharge through wound of hematometric blood 12 days (coincident with menstrual period). At end of 36 days new vagina dissected up to uterus.	Recovery.	Abdominal wound healed on 23d day. Narrow (unsatisfactory?) vagina.
56.	L. Landau (Case II).	*Berl. Klin. Woch.*, 1901, xxxviii, p. 205.	Congenital atresia of cervix (portio absent); hematometra; double hematosalpinx; hemorrhagic ovarian cystoma (right); cyst (size of cherry) in left ovary.	Laparotomy. Double salpingo-öophorectomy. Three months later uterus opened per vaginam giving exit to 3 tablespoonfuls of black blood.	Recovery.	Thinks the hematometra was the result of post operative uterine bleeding of menstrual character.

PRIMARY BLOOD-RETENTION IN SINGLE CANAL.—*Continued.*

No.	Reporter or Operator.	Reference.	Diagnosis.	Treatment.	Result.	Remarks.
57.	F. Villar.	*Rev. Mens. de Gyn. Obst. et Paed. Bordeaux*, 1902, iv, p. 193.	Absence of vagina (lower ¾); hematocolpos; hematometra.	Failure of attempt to reach blood collection per vaginam. Laparotomy. Right salpingo-oophorectomy (for congested tube and cystic ovary). Left adnexa not removed. Total hysterectomy. Vaginal cul-de-sac, cauterized and sewed over. Appendectomy. Closure without drainage.	Recovery.	
58.	Batut.	*Le Bull. Med.* 1902, xvi, p. 587.	Complete absence of vagina (congenital); hematocolpos; hematometra.	After vaginal section blood was emptied but uterus continued latero-flexed.	Recovery.	
59.	F. D'Erchia.	*Arch. di Ost. e Ginec.*, 1902, ix, p. 432.	Absence of hymen; absent vagina (cul-de-sac 2 cm.); hematocolpos; hematometra.	Transverse vaginal incision; blunt dissection to retention tumor; opening made with blunt bistory; edges sutured.	Recovery.	
60.	Langsdorff.	*Zent. f. Gyn.*, 1902, xxvi, p. 660.	Atresia vaginæ; hematocolpos.	Vaginal puncture.	Recovery.	
61.	Guillet (Picqué).	*Bull. et mem. soc. de chir. de Paris*, 1902, xxviii, p. 782.	Complete absence of hymen and vagina; hematometra.	Transverse vaginal incision. Puncture of retention tumor followed by enlargement with scissors. Flaps sutured.	Recovery.	
62.	Fontonnont (Chaput).	*Ibid.*, p. 742.	Absence of vagina; hematometra; hydrosalpinx.	Section of vagina until uterus reached, which was emptied partly with aid of curette Four days later fluctuating right-sided tumor discovered. This was punctured and evacuated.	Recovery.	The formation of the hydrosalpinx was assumed to have been caused by the retention of mucous after the uterus had been emptied (?)
63.	F. Campione.	*Arch. di ost. e ginec.*, 1903, x, p. 329.	Absence of hymen and lower ⅔ of vagina; hematocolpos; hematometra (?)	Transverse sub-urethral section; dissection with finger and knife up to retention tumor. 700 grammes thick blood evacuation.	Recovery.	Normal menses since.
64.	Bouchardiere.	*Ind. Med. Rec.*, Calcutta, 1903, xxiv, 472. *Ind. Med.*, 1903.	Occlusion of vagina.			

PRIMARY BLOOD-RETENTION IN SINGLE CANAL.—*Continued.*

No.	Reporter or Operator.	Reference.	Diagnosis.	Treatment.	Result	Remarks.
65.	Oui.	*Comptes Rend. de la Soc. d' obst. de Gyn. et de Paed. de Paris*, 1903, V, p. 10.	Complete absence of vagina; hematometra; hematosalpinx (left).	Vaginal path dug up to cervix with finger; postero-lateral vulvar incision; membrane obstructing cervix divided; cervix opened with uterine dilator. Much discharge. Tampon. Suture of vulvar incision. Recontraction through neglect. Peritonitic attack 6 months later. Laparotomy. Hematometra and hematosalpinx removed by supra-vaginal hysterectomy. Cervical stump cauterized. Drainage from below; closure of peritoneal cavity.	Recovery.	
66.	J. A. M. Moullin.	*Med. Press & Circular*, 1903 N. S., lxxv, p. 637.	Complete absence of vagina and left appendages; hematocolpos; hematometra. Later pyometra and right tubo-ovarian abscess.	Transverse incision in posterior fourchette; blunt dissection to a depth of 2 or 3 inches. Retention cyst tapped with trocar. Large quantity of retained blood. Drainage-tube. Opening closed 3 months later. Laparotomy. Right tubo-ovarian abscess. Supra-vaginal hysterectomy with gush of foul-smelling pus from cervical canal. Old vaginal tract reopened and drainage-tube inserted. Stump sewed over and peritoneum closed. Patient succumbed 3 weeks later.	Death.	Moullin says: "There is no reason why, had hysterectomy been done in the first instance, the patient should not have made a good recovery."
67.	Gustav Rauscher (Zweifel).	*Ztsch. f. Gebtsh. u. Gyn.*, 1903, XLIX, p. 417.	Atresia vaginæ (upper portion); small hematocolpos; hematometra double hematosalpinx.	Laparotomy; left salpingo-öophorectomy; right salpingotomy. Uterus split at fundus; emptied and wiped dry. On dressing forceps passed into uterine cavity vagina opened up. Uterine incision. Suture of mucosa about cervix to that below. Tampon of Douglas to control hemorrhage. Intra-uterine stem.	Recovery.	Femoral phlebitis during convalescence.
68.	Heidenhain.	*Deutsch. Med. Woch.*, 1904, xxx, p. 1014.	Congenital atresia vaginæ (low); hematocolpos; hematometra; left hematosalpinx and later right hematosalpinx.	Crucial incision, paring off the four angles. Evacuation of retained blood. After 14 days left-sided adherent abdominal tumor (hematosalpinx) discovered. Probatory puncture followed by incision. Same condition, 13 days later on right side. Abdominal incision with intestinal injury and fistula as result. Intestinal resection later. Patient cured but left with ventral hernia.	Recovery.	Menstruating regularly since operation (nine months previously).

PRIMARY BLOOD-RETENTION IN SINGLE CANAL.—*Continued.*

No.	Reporter or Operator.	Reference.	Diagnosis.	Treatment.	Result.	Remarks.
69.	M. Hofmeier.	*Ztsch. f. Gebtr. u. Gyn.*, 1904, lii, p. 1.	Atresia vaginæ (upper ¾); hymen present; hematotrachelos; hematometra.	Dissection upwards in rudimentary vagina to distance of 4 to 5 cm. Laparotomy. Peritoneum split transversely in front of globular (distended) cervix and bladder separated. Cervix incised with evacuation of ½ ounce thick stringy blood. Finger forced into peritoneal cavity through vagina; cervix edges secured in long sutures which were passed down through vagina and vulva. Abdominal wounds closed below and above. Cervix sutured to rudimentary vagina with thick catheter passed to fundus.	Recovery.	This is one of the very few cases on record of nearly complete atresia of the vagina with normally developed and functionating internal genitals, resembling the case reported by me in this paper.
70.	Picqué (P. Batigne).	*Bul. et mem. de la Soc. de Chir. de Paris*, 1904, xxx, 824.	Atresia vaginæ (lower ¾); hematocolpos; hem atometra; left hematosalpinx.	Laparotomy. Left hematosalpinx seen and removed. Right adnexa normal. Uterus, cornu and upper third of vagina distended with ½ litre blood. Supra-vaginal hysterectomy.	Recovery.	Tumor felt like a multilocular ovarian cyst.
71.	G. G. Eitel.	*Northwest Lancet*, 1904, xxiv, p. 406.	Absent hymen; atresia vaginæ (lower ¾); hematocolpos (upper portion); hematometra; right tubo-ovarian hemato-cyst. Left hematosalpinx. Left ovary micro-cystic.	After running temperature for several weeks laparotomy. Right salpingo-öophorectomy. Left salpingostomy, bloody contents pressed out, and incision closed with fine catgut. Several cysts in left ovary punctured. Ventro-suspension. Blunt dissection in perineal region to depth of 1½ inches opening into blood sacs of vagina and uterus. Gauze-vagino-uterine drainage. Secondary laparotomy shortly after with discharge of large amount of cloudy serum.	Recovery.	The doctor writes me (Feb., 1908): "The patient became, as far as any one can tell, entirely well."
72.	G. B. Norberg.	*Kansas City Med. Index Lancet*, 1905, xxvi, p. 423.	Congenital occlusion of cervix uteri; right thickened Fallopian tube (hematosalpinx?).	Cervix dilated and kept open with gauze tent. Menses not properly established for 4 or 5 months; vicarious hemorrhages continued up to this time.	Recovery.	Vicarious tonsillar hemorrhages. Later pregnancy and normal delivery.
73.	P. Horrocks.	*Lancet*, 1905, I, p. 1247.	Imperforate hymen; hematocolpos; hematometra; right hemato-(pyo) salpinx.	Tiny puncture in hymen under cocain; sapremia. A week later free incision followed by irrigation. Septic peritonitis. Enlargement of hymeneal incision followed by laparotomy. Lower abdomen contained foul-smelling pus. Distended right Fallopian tube. Left tube normal. Right salpingotomy. Flushing peritoneal cavity leaving glass drainage-tube.	Recovery.	Stormy convalescence 20 years previously lost a case after similar puncture of distended hymen from infected menstrual blood being driven along the Fallopian tubes into peritoneal cavity.

PRIMARY BLOOD-RETENTION IN SINGLE CANAL.—*Continued.*

No.	Reporter or Operator.	Reference	Diagnosis.	Treatment.	Result.	Remarks.
74.	De Sa Freire.	*Rev. da Soc. de med. e cirurg.* Rio de Janeiro, 1905, IX, p. 121; *Index Medicus*, 1905.	Hemato-colpometra.			
75.	A. Delétrez.	*Bul. Soc. Belge de Gyn. et d' obst. Brux.*, 1906-7 xvii, p 76.	No bulging hymen; vagina absent at lower $\frac{1}{3}$; hematocolpos; hematometra.	Puncture with bistory giving exit to chocolate-colored liquid. Dilatation after 15 days.	Recovery.	Conditions normal 3 months later.
76.	S. Houzel.	*Jour. med. de Brux.*, 1906, xi, 124.	Imperforated hymen; hematocolpos; hematometra.	Punctured and excised the occluding membrane. In one sitting thoroughly emptied the blood-channel.	Recovery.	
77.	J. J. Cox and W. E. Fothergill.	*Br. Jour. of Children's Diseases*, 1906, iii, p. 145.	Partial atresia vaginæ (lower $\frac{3}{4}$); hematocolpos; hematometra.	Vaginal dissection to depth of index finger failed to reach blood-collection. Subsequently a laparotomy was done. Uterine cavity opened with discharge of large quantity of chocolate-colored liquid. Excepting left ovary uterus and adnexa removed. Appendages "not much damaged." Upper vaginal cul-de-sac opened and connected with artificial vagina below. Peritoneal cavity and abdominal wound closed everywhere. New vagina packed with gauze.	Recovery.	A retention of mucus occurred subsequently from the rudimentary vagina but spontaneously discharged itself.
78.	Crowe.	*St. Mary's Gaz.*, London, 1906, xii, p. 103.	Absence of lower part of vagina; hematocolpos; hematometra.			
79.	A. R. Hughes.	*Am. Jour. of Clinical Medicine*, 1907, xiv, p. 753.	Imperforate hymen; hematocolpos; hematometra.	Incision.	Recovery.	At end of 8 weeks uterus reduced to normasize.
80.	Fehling (Case II).	*Monatsch. f. Gebts. u. Gyn.*, 1907, xxv, p. 123.	Complete vaginal atresia; hematometra; hematocolpos; tubes and ovaries small.	Vaginal dissection up to uterus. Lower portion of uterus incised with evacuation of about 40 cc. blood. Mucosa of uterus sutured to external wall to secure a permanent opening. Plastic operation below uniting vulva with lower opening of uterus. New vagina measured 6 to 7 cm.	Recovery.	Normal menstruation on 27th day. Several months later died of bronchitis and catarrhal pneumonia.

PRIMARY BLOOD-RETENTION IN DOUBLE CANAL.

No.	Reporter or Operator.	Reference.	Diagnosis.	Treatment.	Result.	Remarks.
81.	G. Leopold (1883) (Kehrer).	*Das Nebenhorn des dop. Uterus* von E. Kehrer, Heidelberg, 1900.	Hematometra in rudimentary accessory horn (right); hematosalpinx.	Laparotomy (exploratory). Later double salpingo-öophorectomy (second laparotomy 7 months after first).	Recovery.	Had had one child.
82.	Drake-Brockman.	*Br. M. J.*, 1893, I, 1220.	Retention of menses 7 years; atresia hymenalis; septate uterus; hematocolpos only?	Incision with blood continuing to ooze during a half hour.	Death 26 days after operation.	No post mortem examination.
83.	C. J. Cullingworth.	*Trans. Am. Gyn. Soc.*, 1893, xviii, p. 435.	Unilateral atresia of right half of cervix in a case of bicornate uterus with a common cervix; hemato-pyo-metra	Exploratory laparotomy for diagnosis. Closure of abdominal wound. Vaginal incision into swelling. Later division of cervical septum.	Recovery.	
84.	J. E. Burton.	*Liverpool Med. Chir. Jour.*, 1894, p. 14.	Uterus bicornis. Right hematometra and hematosalpinx.	Laparotomy. Right salpingotomy. Distended right horn of a uterus bicornis noted. Three weeks later puncture of hematometra per vaginum.		Was prepared to subsequently cut out a piece of uterus in case of refilling of sac. Not observed longer than 7 weeks. Conditions were favorable for a relapse.
85.	R. Werth.	*Arch. f. Gyn.*, 1894, xlviii, p. 423.	Hematometra in closed imperfectly developed half of a uterus bilocularis.	Laparotomy. Exsection of occluded horn containing retained menstrual blood.	Recovery.	Nagel says this may have been a Wolffian cyst.
86.	H. Löhlein.	*Zent. f. Gyn.*, 1894, xviii, p. 997.	Uterus didelphys; hematometra et hematosalpinx sinistri lateris.	Laparotomy. Blood-stained ascitic fluid in abdominal cavity. Salpingotomy. Amputation of corpus uteri dextri (elastic ligation —double tier of sutures). Drained. Four weeks later lower portion of uterus left at time of laparotomy was incised per vaginum and its edges sewed to keep it from closing.	Recovery.	The hematosalpinx was larger than a child's head, distended with dark blood, and adherent to intestine and appendix. Patient menstruated normally after second operation.
87.	X. O. Werder.	*J. Am. Med. Assn.*, 1894, xxiii, p. 234.	Uterus didelphys; right hematocolpos, hematometra and hematosalpinx.	Laparotomy. Right hematometra punctured, evacuating a quart of tarry fluid; right salpingotomy. Through uterine incision a counter opening made into vagina, gauze passed through wounds in uterus and abdominal wall closed. A month later vaginal sac freely incised. Septum between vagina completely excised and edges sutured.	Recovery.	Hematosalpinx was 6 inches long and 4½ in. in circumference. One fundus was removed 3 inches from the second. Lower end of the uterine tumor consisted of an incomplete vagina (right side); left vagina normal.

PRIMARY BLOOD-RETENTION IN DOUBLE CANAL.—*Continued.*

No.	Reporter or Operator.	Reference.	Diagnosis.	Treatment.	Result.	Remarks.
88.	Skalkowski (1894) (Kehrer).	*Das Nebenhorn des dop. Uterus* von E. Kehrer, Heidelberg, 1900.	Rudimentary horn with double uterus. Hematosalpinx.	Laparotomy.	Recovery.	
89.	Edmond Blanc.	*La Loire Medicale*, 1895, p. 3.	Unilateral hematometra in uterus bipartitus.	With "trocar-sound" punctured left sided uterus (cervix absent) and dilated opening.	Recovery.	
90.	Sänger (1896) (Kehrer).	*Das Nebenhorn des dop. Uterus* von E. Kehrer, Heidelberg, 1900.	Hematometra in rudimentary accessory horn (left); hematosalpinx.	Laparotomy. Double salpingo-oöphorectomy.	Recovery.	
91.	Rossa (1896) (Kehrer)	do.	Left hematometra in rudimentary horn of double uterus; hematosalpinx.	Puncture hematosalpinx; then extirpation of hematometra and hematosalpinx.	Recovery.	Peritonitic adhesions about both blood-sacs.
92.	B. S. Schultze—H. Zillessem (Kehrer).	*Das Nebenhorn des. dop Uterus* von E. Kehrer, Heidelberg, 1900.	Hematometra (left) in uterus septus bilocularis; no hematosalpinx.	Laparotomy. Extirpation of sac.	Recovery.	
93.	A. H. N. Lewes.	*Br. Med. J.*, 1896, ii, p. 1131.	Complete absence of vagina. Double uterus with double hematometra.	Vaginal operation revealed a double uterus, each half distended with retained menstrual blood. Evacuation.	Recovery.	Vagina kept open with ring pessary. Eight months later patient well and menstruating regularly. Pessary had to be removed under anesthesia and regretted not having done a laparotomy at the start. Very rare case.
94.	Mackenrodt.	*Ztsch. f. Gebts. u. Gyn.*, 1896, xxxv, 302.	Uterus didelphys; hematometra in occluded half.	An attempt a year previously to remove the little occluded uterus by a vaginal operation had failed. A second effort was successfully made by the same route. By the aid of the Pæquelin cautery the second uterus (filled with retained blood) was amputated.	Recovery.	Spurting hemorrhage in spite of the cautery.

PRIMARY BLOOD-RETENTION IN DOUBLE CANAL.—*Continued.*

No.	Reporter or Operator.	Reference.	Diagnosis.	Treatment.	Result.	Remarks.
95.	A. Calmann (Rosthorn).	*Zent. f. Gyn.*, 1896, XX, p. 598.	Uterus unicornis cum cornu rudim entari dextro; no cervix (right); hematosalpinx (right); hematometra in rudimentary horn.	Laparotomy. Right salpingo-öophorectomy. Several months later amputation of right horn of uterus.	Recovery.	
96.	E. Lauwers	*Bul. de l'acad. Roy. de Medicine*, 1896, X, p. 570.	Right uterus unicornis; left rudimentary horn with hematometra. Complete atresia vaginæ; hematosalpinx.	Blunt vaginal dissection; incision of tumor on aspirating needle as guide. Large amount of liquid escaped. Injury to bladder and peritoneum recognized. Immediate laparotomy. Free blood present in abdominal cavity. Amputation of left rudimentary uterus with sac of hematosalpinx. Vaginal and abdominal drainage.	Recovery.	
97.	E. Schwartz.	*Rev. de Gyn. et de Chir. abd.*, 1897, I, 961.	Congenital absence of lower $\frac{2}{3}$ of vagina; hematocolpos; left cornu normal; undeveloped right cornu.	Exploratory laparotomy. Blunt vaginal dissection with puncture of liquid tumor giving exit to thick, viscous yellow liquid.	Recovery.	
98.	Barone.	*Ind. Med.*, 1897.	Uterus didelphys; atresia of one side; hematometra; hematosalpinx.			
99.	F. H. Maier.	*Am. Gyn. and Obst. J.*, 1897, xi, p. 713.	Complete absence of vagina; unicornate uterus; left tubo-ovarian blood tumor.	Vaginal canal dissected out. Laparotomy. Adhesions of (tubo-ovarian) tumor to surrounding parts. Filled with dark clotted and fluid blood. Salpingo-öophorectomy. Vagina kept open artificially.	Recovery.	No horn tube or ovary on right side.
100.	A. L. Galabin.	*Practitioner*, 1897, lviii, p. 351.	Left hematosalpinx; left hematometra in imperfectly developed uterus (no cervix).	Laparotomy. Blood-distended left Fallopian tube removed. Left uterus (with 1 oz. bloody contents) amputated.	Recovery.	
101.	J. L. Lackie.	*Edinb. Med. J.*, 1897, I, 274.	Right hematometra in double uterus (unicollis).	Probatory diagnostic aspiration. Later incision of sac. Destruction of partition between two uterine cavities. Original incision sutured. Drainage tube in cervical canal.	Recovery from operation. 4/12 later death from other causes.	Menstruated every two weeks alternately from each uterine half during four months. Then had attack of rheumatism with cardiac complications from which she died.

PRIMARY BLOOD-RETENTION IN DOUBLE CANAL.—*Continued.*

No.	Reporter or Operator.	Reference.	Diagnosis.	Treatment.	Result.	Remarks.
102.	Jos. Godart	*Bul. Soc. Belge de Gyn. et d' obst.*, 1898-9, IX, p. 14.	Complete absence of vagina. Right uterus unicornis; left rudimentary horn. Pelvic hematocele at outer end of tube.	Blunt dissection of perineum to depth of 7 cm. 30 grammes blood evacuated. Peritoneal cavity accidentally opened and bladder injury. Parts sutured. Several weeks later abdominal panhysterectomy. Fresh injury to bladder repaired.	Recovery.	Well after one year.
103.	F. Kleinhals.	*Monat. f. Gebts u. Gyn.*, 1897, V, p. 231.	Congenital atresia of left half of uterus bilocularis; left hematometra and hematosalpinx.	After probatory puncture trocar draws off 250 cc. blood; 9 days later laparotomy. Left salpingo-öophorectomy.	Recovery.	As hematosalpinx did not disappear after emptying the hematometra laparotomy was decided on.
104.	Lambret.	*Bul. Med.*, 1898, xii, p. 133.	Hematometra in rudimentary horn (right) of uterus unicornis.	Laparotomy. Amputation of small blood-filled rudimentary horn with rudimentary tube and ovary (right side).	Recovery.	
105.	Mainzer (L. Landau).	*Arch. f. Gyn.*, 1898, LVII, p. 681.	Double uterus with atresia of left side; left hematometra; left hematosalpinx.	Probatory vaginal puncture into left hematometra. Laparotomy to inspect conditions present. Left hematometra incised and emptied per vaginam. Left salpingo-öophorectomy.	Recovery.	Menses subsequently normal.
106.	J. v. Rosenthal.	*Mon. f. Gebts. u. Gyn.*, 1898, viii, p. 273.	Uterus bicornis; hematometra in right horn continuous with hematotrachelos.	Several years previously spontaneous rupture of labial abscess. Vaginal tumor aspirated (1 litre coffee-colored liquid). A week later foul-smelling discharge with evidence of septic infection. Supra vaginal hysterectomy removing both uteri and adnexa. The 6 to 7 cm. deep sac was drained into vagina and washed out daily. Abdominal wound infection.	Recovery.	
107.	Vignard.	*Gaz. med. de Nantes*, 1899, xvii, p. 83.	Bifid uterus; lateral hematometra and hematosalpinx (right).	Laparotomy. Right salpingo-öophorectomy Abdomen closed. Vaginal section of right hematometra. Flood of liquid pours out from cavity 15 cm. deep. Suture of edges of wound and drainage.	Recovery.	Cavity of right uterus 6 cm. deep 11 weeks later. The blood-filled Fallopian tube presents an obliterated uterine end.
108.	Witthauer.	*Mon. f. Gebts. u. Gyn.*, 1899, IX, p. 205.	Double uterus; left hematosalpinx; hematometra.	Laparotomy. Left salpingotomy. Right uterus smaller with normal tube and ovary. Left uterus distended. Abdomen closed. Vaginal incision into left uterus with expulsion of 1 litre of blood. Suture of edges of wound; thick drainage tube left in uterus.	Recovery.	A left accessory Fallopian tube was discovered in this case. Intercurrent post-operative pneumonia.

PRIMARY BLOOD-RETENTION IN DOUBLE CANAL.—*Continued.*

No.	Reporter or Operator.	Reference.	Diagnosis.	Treatment.	Result.	Remarks.
109.	O. A. Boije (Engström).	*Mitth. aus d. Gyn. Klin.* (Engström), 1899-1901), iii, p. 225.	Uterus bicornis unicollis. Hematometra in right rudimentary horn.	Laparotomy. Amputation of rudimentary horn. Both tubes normal.	Recovery	
110.	A. Cocchi (Pestalozza).	*An. di ost e Ginec.*, 1900, xxii, p. 386.	Uterus didelphys; right hematometra; right hematosalpinx.	Extirpation of right uterus and adnexa. Reconstruction of right pelvic floor.	Recovery.	
111.	B. H. Wells.	*Am. Jour. of Obst.*, 1900, XLI, 317 (Case III).	Retro-hymeneal atresia; double uterus; double vagina; bilateral hematometra and hematocolpos.	Crucial incision; flaps removed. Vaginal mucosa and hymeneal edges sutured. One quart of liquid evacuated at once.	Recovery.	
112.	F. W. N. Haultain.	*Br. Med. Jour.*, 1900, I, 1279.	Undeveloped double uterus; hematometra (right).	Laparotomy. Hematometra in right horn had ruptured between folds of broad ligament. No tubal distension. Double oöphorectomy. Amputation of distended horn. Extravasated blood washed out and broad ligament layers sutured.	Recovery.	
113.	C. Jacobs.	*Soc. Belge de Gynec.*, 1900-1901, xi, p. 189.	Uterus didelphys; double vagina (one ending in blind sac); pyocolpos; pyo-trachelos; pyo-metra.	Laparotomy. One atrophied uterus continuous with a normal vagina. Second enlarged uterus terminated in closed vagina at the side. Hysterectomy. Vaginal drainage. Peritoneum closed at pelvic floor over drain. Abdomen closed without drainage.	Recovery.	A vaginal puncture for retained blood had been done 2 years previously. Jacobs feels that this case should have been simply incised and drained per vaginam. Stormy convalescence.
114.	Abel (R. Katz, 1904).	*Bul. Klin. Woch.*, 1901, xxviii, p. 1280.	Double uterus. Hematometra in right atrophied rudimentary horn. Right hematosalpinx.	Vaginal (anterior) section. Puncture of hematosalpinx. Amputation of right horn and tubal sac.	Recovery.	Menses normal six months later.
115.	Labusquiere (Obser. II).	*Ann. de Gyn. et d'Obst.*, 1901, xlvi, p. 141.	Vaginal atresia (right side and congenital). Double uterus. Hematometra of the right cornu and right hematosalpinx and hematovarium.	Double abdominal hysterectomy (Doyen's method). Adhesions to cecum and appendix. Double salpingo-oöphorectomy.	Recovery.	

PRIMARY BLOOD-RETENTION IN DOUBLE CANAL.—*Continued.*

No.	Reporter or Operator.	Reference.	Diagnosis.	Treatment.	Result.	Remarks.
116.	Kézmarszky-Baecker.	*Zent. f. Gyn.*, 1896, p. 883; also *Arch. f. Gyn.*, 1901, LXIV, p. 98.	Uterus didelphys; hematometra; hematosalpinx sinistra.	Left salpingotomy. Amputation of the hematometra with suture of sac to abdominal wound ("herausnähen des sackes"). Died same day of shock.	Died.	Absence of left kidney.
117.	E. Herz.	*Wien. Med. Woch.*, 1901, LII, p. 1101.	Absent vagina. Uterus unicornis sinistra; hematocolpos.	Vagina constructed by blunt dissection which closed up and hematocolpos formed. Obstruction disposed of again. Dilatation.	Recovery.	
118.	Paul Mathes.	*Ztsch. f. Heilk.*, 1900, I, 359.	Uterus bicornis unicollis; hematotrachelos (right side). Rupture of necrotic tubal wall; hematosalpinx.	Vaginal incision and drainage with tube evacuating large quantity brown liquid. A week later sudden cramp. Laparotomy. Bloody pus. Right tube bluish-red convoluted and ruptured. Right corpus uteri small; right cervix dilated to size of apple. Left uterus tube and ovary normal. Radical operation, leaving left ovary to prevent climax precox. Abdominal wall abscess.	Recovery.	Thinks the rupture in the tube was due to (1) a tubal wall weakened in its resisting power; (2) the thinnest portion gave way on account of the diminution in size of the hematotrachelos; and (3) because of the restlessness of the patient.
119.	Pretorius (1899) (Stratz).	*Ztsch. f. Gebts. u. Gyn.*, 1901, XLV, p. 1.	Hematosalpinx (right). Hematometra (right). Double uterus with single cervix. Vagina duplex; congenital right sided atresia.	Laparotomy. Right hematometra and hematosalpinx with normal uterus seen to left. Adhesions to cecum and appendix. Connective tissue adhesions to sacrum behind large right-sided tumor. Double hysterectomy (Doyen's method). Removal of hematosalpinx with moderate-sized hematovarium.	Recovery.	
120.	C. H. Stratz.	*Ibid.*	Double uterus. Right normal. Left hematometra and hematosalpinx with hematovarium (moderate degree).	Laparotomy. Double salpingo-öophorectomy. Right hematometra punctured per vaginam.	Recovery.	Uterine end of hematosalpinx only admitted fine probe. Symptoms persisted for a year and Stratz considers hysterectomy justifiable in similar cases.
121.	Carl V. Pauer (Elischer).	*Zent. f. Gyn.*, 1902, xxvi, p. 660.	Uterus duplex separatus; left hematosalpinx.	Laparotomy. Left salpingo-öophorectomy. Amputation of left (rudimentary) uterus. Removal of a tumor from omentum (accessory spleen). Right uterus and adnexa left in situ.	Recovery.	

PRIMARY BLOOD-RETENTION IN DOUBLE CANAL.—*Continued.*

No.	Reporter or Operator.	Reference.	Diagnosis.	Treatment.	Result.	Remarks.
122.	Labusquiere (Observ. III).	*Ann. de Gyn. et d'obst.*, 1901, xlvi, p. 141.	Double uterus; left hematosalpinx; left hematometra.	Laparotomy. Left salpingo-öophorectomy. Hematometra punctured per vaginam.		Uterine end of hematosalpinx impervious to a sound.
123.	E. Marvel.	*Ann. Gyn. and Ped.*, 1903, xvi, p. 625.	Uterus duplex unicollis. Left hematometra double ovarian (hemato?) cysts.	Per vaginam incision into mass to left of cervix evacuating teacup full of gelatinous blood. Laparotomy. Double hysterectomy including respective ovary and tube.	Recovery.	
124.	G. Rauscher (Zweifel).	*Ztsch. f. Gebt. u. Gyn.*, 1903, XLIX, 417.	Absent vagina, lower part; hymen septus present. Uterus duplex; hematometra left horn; left hematosalpinx; hematocolpos.	Laparotomy. Left salpingotomy. Anterior uterine wall incised and blood mopped out. Opening made through uterine cavity to vagina. Uterine wound sutured. Vaginal opening enlarged. Hymen exsected. Vagina stitched to skin.	Recovery.	After 15 months periods regular and local conditions apparently satisfactory.
125.	Brohua (Fraipout).	*Ann. Soc. Med. Chir. de Liege*, 1903, XLII, p. 511.	Uterus didelphys; atresia of cervical portion of left horn; left hematometra.	Laparotomy. Amputation of left horn containing thick blood.	Recovery.	It was thought in this case that in the intermenstrual periods the uterine blood in the shut-off horn was reabsorbed.
126.	C. H. Hare.	*Boston Med. and Surg. J.*, 1903, p. 148.	Uterus bicornis duplex, two cervical canals above, one external os. Stricture of vagina. Dysmenorrhea from obstructed flow of menstrual blood.	Several minor operations from below had met with failure. Laparotomy. Ovaries and tubes normal. Supra-vaginal hysterectomy.	Recovery.	The blood retention was evidently partial in this case.
127.	Gouilloud.	*Bul. de la Soc. de Chir. de Lyon*, 1903, vi, p. 27.	Double uterus. Left hematometra. Left vagina was a closed cul-de-sac.	Laparotomy. Left öophorectomy (ovary normal). Left tube atrophied. Rudimentary, blood-distended left uterus exsected (like a myomectomy). Right uterus and adnexa left.	Recovery.	The exsected uterus showed no communication with the right uterus, vagina or left tube.
128.	Cerné.	*Rev. Med. de Normandie*, 1904, vi, p. 313.	Uterus didelphys unicollis; hematometra dextra with atresia partialis of right uterine opening.	Amputation of right uterus.	Recovery.	Left uterus functionating properly since the operation.

PRIMARY BLOOD-RETENTION IN DOUBLE CANAL.—*Continued.*

No.	Reporter or Operator.	Reference.	Diagnosis.	Treatment.	Result.	Remarks.
129.	G. Gross.	*Ann. de Gyn.*, 1904, I, p. 23.	Uterus bicornis unicollis; hematometra in left accessory rudimentary horn.	Laparotomy. On seizing the rudimentary horn with Museux forceps black blood oozes forth. Enucleation of tumor. Left salpingo-oophorectomy. Suture of uterine and abdominal wounds.	Recovery.	
130.	Dalmon & Mounet (Chaput).	*Bull. de la Soc. Anat. de Paris*, 1904, LXXIX, p. 802.	Imperforation of hymen; atresia lower segment vagina; uterus bicornis; hematocolpos swollen cornua; right hematosalpinx with right tube showing torsion.	Vaginal dissection to depth of 5 or 6 cm. before hematocolpos reached. Upper vaginal cuff sutured to labia. On third day fever with thready pulse. Mass felt in rectum punctured. Died a few hours later.	Death.	At autopsy found that right hematosalpinx had been opened into by the vaginal section. Pedicle twisted. No cervix found. No peritonitis. Attributes death to "reflex intestinal occlusion" due to torsion of the tube (Chaput).
131.	Elis Essen-Moller.	*Nord. Med. Arkiv.*, 1904, Afd. I, Häft 2, No. 7, p. 1.	Uterus bicornis; hematometra in right rudimentary horn (ruptured).	Laparotomy. Free clots of blood with evidences of peritonitis in abdominal cavity. Tubes and ovaries normal. Amputation of accessory horn leaving right ovary and most of right tube.	Recovery.	The exsected uterus was distended with black blood and was the seat of a spontaneous perforation.
132.	Lindquist.	*Zent. f. Gyn.*, 1904, xxviii, p. 395.	Uterus bicornis; hematometra cornu dextri; cervix normal with rudimentary horn attached to right.	Laparotomy. Left ovary cystic, tube short. Left salpingo-oophorectomy. Egg-sized body seen on right side to which rudimentary tube and wound ligament attached. Right ovary not found. Exsection of rudimentary uterus. Vaginal drainage. Dermoid cyst of right ovary later found in Douglas' sac and removed.	Recovery.	Nephropexy (right) 10 years previously. Cavity of rudimentary uterus the size of a bean.
133.	A. Prima Pintor.	*Ibid.*, p. 407.	Uterus bicornis; hematometra in right rudimentary horn. Cervix normal.	Anterior colpotomy. Left adnexa normal. Right adnexa degenerated and atrophic. Vaginal hysterectomy leaving left adnexa.	Recovery.	Gave birth to 2 children. Mistaken for an adnexal tumor or a fibromyoma.
134.	M. Hofmeier.	*Ztsch. f. Gebts. u Gyn.*, 1904, lii, p. 1.	Double uterus; hematometra left horn; later left heamtotrachelos and hematometra, and 3-months' pregnancy in right uterus.	Laparotomy. Removal of tumor filled with thick cheesy blood (left horn). Following year left uterus and cervix became so much distended with blood as to require broad incision (per vaginam) allowing ½ litre smeary blood to escape. Five days later an overlooked 3 months' ovum was expelled from the right uterus.	Recovery.	

PRIMARY BLOOD-RETENTION IN DOUBLE CANAL.—*Continued.*

No.	Reporter or Operator.	Reference.	Diagnosis.	Treatment.	Result.	Remarks.
135.	Le Bec.	*Rev. de Gyn. et de Chir. abd.*, 1904, viii, p. 387.	Uterus didelphys. Left hematometra shut off from right uterus and vagina. Right ovarian hematocyst. Left hematosalpinx and ovarian hemato-cyst (both adherent).	Panhysterectomy.	Recovery.	Patient had given birth to one child 7 years previously.
136.	Prochownick.	*Munch. Med. Woch.*, 1904, LI, 233.	Uterus bicornis; hematometra in right rudimentary accessory horn; right hematosalpinx (twisted 180).	Laparotomy. Left-sided genital apparatus normal. Right rudimentary horn; size of walnut, filled with blood. Slightly blood-filled right tube twisted in itself up to club-shaped fimbricated end which is quite distended. Right ovary normal. Amputation of rudimentary horn and hematosalpinx.	Recovery.	Earliest case for operation of its kind on record. Thinks his case proves the etiology of pure mechanical conditions as causing hematosalpinx (no inflammatory or infection antecedents).
137.	A. Pellegrini.	*La Ginecologia* 1904, I, 709.	Double uterus and double vagina. Right hematometra. Right hematosalpinx. Right hematic cystic ovary. Left uterus, cervix, vagina, tube and ovary normal. Congenital absence of right kidney and ureter. Fatty degeneration of liver.	Laparotomy. Incision of mass with evacuation of 1000 grammes black thick plood. Isolation and extirpation of most of the sac. Sub-normal temperature and weak pulse. Death 4 days later.	Death.	Mistaken for a fibromyoma of the uterus.
138.	N. J. A. F. Boerma.	*Ned. Tijd. voor Verlosk en Gyn.*, 1904, p. 299.	Uterus bicornis duplex. Hematometra dextra.	Exploratory vaginal puncture into tumor with negative (bacteriological) findings. Laparotomy. Tumor in right horn of a uterus duplex size of child's head. Right adnexa not enlarged but closely adherent to cornu. Left horn broadly united to tumor with normal tube and ovary. Total extirpation of uterus with the adherent adnexa, opening into vagina which is later sewed over.	Recovery.	
139.	A. Doran.	*Jour. Obst. & Gyn. of Brit. Empire*, 1905, vii, p. 167.	Uterus septus unicollis (fibroid tumor in the septum); left hematosalpinx; left ovarian hematocyst.	Laparotomy. Supra-vaginal hysterectomy with removal of both tubes and ovaries.	Recovery.	Uterus was nearly spherical in shape and extended up to the umbilicus.
140.	C. S. MacGreggor.	*Glasgow Med. Jour.*, 1906, LXV, p. 340.	Imperforate hymen; hematocolpos; hematometra sinistra.	Incision of hymen; discharge of 2 pints of blood; double-horned uterus now mapped out clearly.	Recovery.	

PRIMARY BLOOD-RETENTION IN DOUBLE CANAL.—*Continued.*

No.	Reporter or Operator.	Reference.	Diagnosis.	Treatment.	Result.	Remarks.
141.	Quénu et Le Sourd.	*Rev. de Chir.*, 1906, XXXIII, p. 1.	Uterus duplex unicollis; right hematometra.	Laparotomy. Evidences of blood in omentum and peritoneal cavity. Right supravaginal hysterectomy. Right salpingo-oophorectomy. Nine months later incision into cervical stump distended with retained blood on right side. Suture of this cavity to vaginal mucosa.	Recovery.	Case was first diagnosticated as tubercular salpingitis bilateralis.
142.	J. E. Walker.	*Am. Jour. Surg.*, 1906, XX, p. 33.	Complete absence of vagina; double uterus. Right ovary three times normal size with three hematomata.	Exploratory laparotomy. Right tube normal and attached to rudimentary right uterus. Right hematoma of ovary. Left uterus same size. Ovary and tube fairly normal. Double total hysterectomy.	Recovery.	The history indicates the possibility of a spontaneous rupture of a pelvic hematocele into the rectum with the discharge of "about a quart of blood."
143.	M. Gauthier.	*Bull. et mem. de la Soc. Anat. de Paris*, 1906, LXXXI, 570.	Typical uterus didelphys; each uterus having its own vagina and adnexa. Transverse septum in upper part of vagina leading to small cervix and uterus. To the left a distended cervix and uterus forming a large (blood) cyst, one of its tubes being a hematosalpinx; other ovary seat of a blood cyst.	Total abdominal hysterectomy.	Recovery.	Had suffered from painful menstruation and diagnosis of adnexal cyst had been made.
144.	H. N. Vineberg.	*Am. Jour. of Obst.*, 1907, LV, p. 843.	Uterus didelphys; right hematocolpos. Complete atresia of right cervical os.	Laparotomy. Two normally developed cornua, right being larger. From cervix a large globular tumor on right side seen in pelvis. Bladder separated and ovarian ligament tied off. Incision into tumor with discharge of black tarry blood. Total hysterectomy.	Recovery.	Mistaken for a fibroid growth in the cervix.
145.	E. Rosenfeld (Hahn).	*Munch. Med. Woch.*, 1907, LIV, p. 297.	Uterus duplex; vagina duplex; atresia vaginæ sinistræ; left hematocolpos.	Exploratory laparotomy showing two normal uteri united by a broad connective tissue bridge, left uterus with its tube and ovary pushed upwards by the large vaginal tumor. Abdomen closed. Left atresia vaginæ opened from below evacuating dark blood and partly yellow, foul-smelling liquid. Subsequently the vaginal septum was excised.	Recovery.	Soon afterwards became pregnant on the side operated (left), and had a normal delivery.

SECONDARY BLOOD-RETENTION IN SINGLE CANAL.

No.	Reporter or Operator.	Reference.	Diagnosis.	Treatment.	Result.	Remarks.
146.	H. A. Kelly (Robb)	*Johns Hopkins Reports*, 1892-4, iii, p. 429.	Atresia vaginæ; hematocolpos; hematometra.	Opened with trocar and dilator and restoration of vaginal canal (500 cc. retained menses).	Recovery.	Instrumental delivery 8 years previously.
47.	Paul Petit.	*Cong. Franc de Chirng.*, 1892, p. 633.	Primary vaginal stenosis; secondary cicatricial atresia of vagina and cervix. Hematocolpos et hematometra. Pelvic abscess.	Dilatation of strictured area (5 or 6 cm. from vulva) with discharge of coffee-colored blood and clots. Right broad ligament abscess. Laparotomy. 1½ litres of pus evacuated. Right tube fused to sac. Exsection ⅔ sac; marsupialization of the rest with drainage. Vaginal stricture later divided bilaterally.	Recovery.	Dystocia due to primary vaginal stenosis requiring incision followed by forceps delivery. Subsequent cicatricial closure of vagina (nearly complete).
148.	S. Buongiorno.	*Il Progres Med.*, 1892, V, p. 628.	Complete cervical atresia with hematometra; obliteration of external os.	Artificial reopening of cervical canal.	Recovery.	30 months after last confinement (craniotomy).
149.	Lewolff (1893) (Neugebauer).	*Wratsch.*, 1893, No. 28; *Ann. de Gyn.*, 1895, p. 190.	Vaginal atresia; hematometra; hematosalpinx sinistra.	Attempt to reach blood accumulation per vaginam abandoned. Two months later peritonitis. Laparotomy. Left salpingo-oophorectomy. Right oophorectomy. Uterus and right tube buried in adhesions. Late discharge of pus from vaginal cicatrix.	Recovery.	Gangrene of vagina and vulva after typhus
150.	J. D. Doorman.	*Ned. Tij. voor Verlosk. en Gyn.*, 1893, IV, p. 16.	Vaginitis adhesiva; hematocolpos (hematometra?).	Division of obstructing "bridle" causing vaginal occlusion.	Recovery.	"Leucorrhea" 2 years previously.
151.	Kochenburger.	*Ztsch. f. Gebts. u. Gyn.*, 1893, xxvi, p. 60.	Membraneous closure of introitus vaginæ. Hematocolpos; hematotrachelos.	Incision.	Recovery.	One year previously dystocia requiring perforation.
152.	B. C. Hirst.	*Am. Jour. of Obst.*, 1895, xxxii, p. 902.	Hematocolpos due to vaginal atresia.	After confinement double oophorectomy (portion of one ovary left); a year later spontaneous rupture of an hematocolpos into the bladder. Hematocolpos later opened by operation with resulting vesico-vaginal fistula which two operations failed to close. Rectal puncture of blood collection followed by septic infection. Hysterectomy (abdominal). Spontaneous closure of fistula.	Recovery.	After delivery of first child a plastic vaginal operation done which probably accounts for the atresia (in the upper third) of the vagina.

SECONDARY BLOOD-RETENTION IN SINGLE CANAL.—*Continued.*

No.	Reporter or Operator.	Reference.	Diagnosis.	Treatment.	Result.	Remarks.
153.	S. Pozzi.	*Bul. et mem. de la Soc. de Chirng. de Paris*, 1895, xxi, 509.	Cicatricial atresia of cervix. Hematometra; double hematosalpinx; retro-uterine hematocele.	Laparotomy. Removal of adnexa (hematosalpinx-tumors) with pelvic drainage. Unsuccessful attempt to open up uterine canal. Through infection hematometra converted into pyometra. Vaginal hysterectomy 42 days later.	Recovery.	Curettage and amputation of cervix following delivery 6 years previously. Four years later cicatricial obliteration of cervix.
154.	Courant.	*Index Medicus*, 1896.	Acquired atresia tubæ; sacto-salpinx hemorrhagica (hematosalpinx).			
155.	E. I. Persinger.	*Cincin. Med. Jour.*, 1896, xi, No. 2, p. 67.	Traumatic vaginal atresia; hematocolpos.	Puncture with trocar giving exit to a pint of liquid.	Recovery.	After forceps delivery. Well one year after operation.
156.	L. Van der Hoeven.	*Ned. Tij. dschr. v. Verlosk en Gyn.*, 1897, viii, p. 1.	Complete obliteration of vagina; hemato-pyosalpinx.	Laparotomy; ascites; intestinal adhesions too dense to permit of removal of sac which ruptures during operation, allowing escape of foul smelling pus. Gauze packing. Blunt dissection of vagina to cervix. Gauze packing of uterus and vagina. Vagino-rectal fistula which healed spontaneously.	Recovery.	Age 35; married nine years; never pregnant; menses regular from 14th year down to 1½ years previously when she had diphtheria.
157.	P. Hyrup-Pedersen.	*Colorado Med. Jour.*, 1897, iii, p. 9.	Atresia vaginæ; hematocolpos.	After 4-inch separation of agglutinated vaginal surfaces the "thick capsule of tumor" incised giving exit to 1½ quarts bloody liquid. Insertion of tube every night but vagina has a tendency to close up as the patient is still "unmarried."	Recovery.	Three years previously diphtheritic vaginitis (aged 13) during which a cast of vagina was expelled.
158.	Treub.	*Ned. Tij. v. Verlosk en Gyn.*, 1899, X, 305.	Atresia vaginæ. Left hematovarium; salpingitis (interstitial?). Hematometra.	Laparotomy, for presumed left hematosalpinx. Enlarged ovary with bloody contents found instead and removed. Tube thickened. Attempt to reach the uterus from below per vaginam failed. Second laparotomy with removal of uterus. Numerous adhesions.	Recovery.	Two years previously had been ordered vaginal injections of potassium permanganate (vagina patent and uterus normal at the time). Uterus was thick-walled with small blood-filled cavity.
159.	Witthauer.	*Monats. f. Gebts. u. Gyn.*, 1899, IX, 205.	Cervical atresia and vaginal obliteration.	Blunt dissection of vaginal cicatricial tissue; cervical section with suture of opening and gauze packing; uterus contained 2 litres of tarry liquid.	Recovery.	After third confinement (normal), 5 years previously, was ordered vaginal douches for a purulent discharge, after which menses ceased.

SECONDARY BLOOD-RETENTION IN SINGLE CANAL.—*Continued.*

No.	Reporter or Operator.	Reference.	Diagnosis.	Treatment.	Result.	Remarks.
160.	Wm. Gardner.	*Mont. Med. Jour.*, 1900, XXIX, p. 58.	Occlusion of cervix due to adhesive inflammation.	Supra-vaginal hysterectomy. Uterus size of adult head and blood spurted upwards to a distance of two feet.	Recovery.	Woman, multipara, aged 51, menopause 2 years. Cup of prolapsus pessary caused cervical obliteration and hematometra (which was diagnosticated as fibroid tumor, or solid tumor of ovary or possible malignant neoplasm).
161.	T. G. Chrysopathes (Schwartz-Halle).	*Ztsch. f. Gebts. u. Gyn.*, 1901, XLIV, p. 238.	Occlusion of uterine ends of both tubes by adenomyomata; double hematosalpinx.	Laparotomy. Right hematosalpinx size of fist; smaller one on left side. Double salpingo-oophorectomy. Abdomen closed. Immediate vaginal hysterectomy with drainage per vaginam.	Recovery.	The neoplasm had produced occlusion of uterine ends of both tubes; the hematosalpinx was the result of blood directly exuded by the tubal mucosa.
162.	J. La Bouchardiere.	*Ind. Med. Record*, 1903, XXIV, p. 472.	Occlusion of vagina; hematocolpos; hematometra.	By the use of blunt dissection and the knife the vaginal canal was reopened and two pounds of tarry liquid evacuated.	Recovery.	After a 5-days' labor with impaction of fetal head vaginal obliteration followed, and menses were retained for about one year.
163.	H. Violet and G. Cotte.	*Lyon Medical* 1907, cviii, p. 690.	Congenital stricture of vagina; post-partum vaginal occlusion; hematocolpos.	Dystocia due to congenital vaginal occlusion at depth of 3 cm. No opening could be found large enough to pass a probe. Incision of septum followed by spontaneous delivery. A year later complete closure of vagina by cicatricial tissue. Incision permitted escape of thick black non-coagulated blood.	Recovery.	
164.	A. Henderson.	*Br. Med. J.*, 1907, I, 138.	Vaginal atresia; hematocolpos.	Spontaneous rupture and discharge of blood. Easy operation to restore vaginal lumen.	Recovery.	Five children; last one a year previously, non-instrumental; three months later dyspareunia.

CYSTOSCOPY: ITS RELATION TO THE SEPARATION OF THE URINE AND INTRAVESICAL MANIPULATION.

BY FOLLEN CABOT, M.D.

Professor of Venereal and Genito-Urinary Surgery, New York Post-Graduate Medical School and Hospital.

Several instruments other than cystoscopes have been devised with the idea of temporarily dividing the bladder into two lateral halves and in this way attempting to keep the urine separate as it comes from each kidney. These so-called *separators* or *segregators* are occasionally useful when dealing with a healthy bladder. This reservation, however, begs the whole question, because we are *never sure* that a given bladder is perfectly normal. We may be right in 98 per cent. of cases, but in the remaining two per cent. we may be mistaken, and so this possible source of error, even in a small percentage of instances, makes the method unreliable. In contrast to the cystoscope and the ureter catheter, this method of artificially dividing the bladder may be called a *blind* one. The only use for an instrument of this character is, in my opinion, in cases of infection of the bladder in consequence of which condition it is undesirable to run any chance of contaminating a healthy ureter and kidney by the introduction of a ureteral catheter. In a case of this type, however, it is necessary to use the cystoscope first in order to determine the condition of the bladder; but with a diseased bladder it would be difficult to exclude it as the origin of pus or blood found in either of the separate urines as collected by one of these segregators. In the presence, however, of elements in the urine necessarily of kidney origin and found on one side only, it is of value. In the other class of cases where its use has been urged, that is, where a ureteral opening cannot be found by aid of the cystoscope, it may also be of service. However, these instances are so rare and there are so many methods in common use for locating an obscure ureteral opening, that in the hands of one with moderate experience the cystoscope and ureter-catheter are the instruments of choice.

Harris, of Chicago, some years ago devised an instrument he called a segregator. With this segregator the bladder is di-

vided into two parts by placing an arm of the instrument in the rectum or vagina, thus forming a watershed between the two ureters. The other arm of the instrument is then introduced into the urethra as we would a sound in the male. This arm is divided into two parts which swing on joints, and, after it is in position in the bladder, it is opened, one-half of the beak going into the cul-de-sac on side made by the watershed and the other half going into the opposite side. These two halves are provided with eyes which connect with the lumen of each half of the shaft of the instrument and drain off urine as it is emptied into the bladder from each kidney. It is certainly an ingenious instrument and easily manipulated. I, however, do not use it for the reasons I have already stated. Downs, of Philadelphia, has devised an instrument on somewhat similar lines, taken from the original model of Harris's with some modification.

The Luys' and Cathélin *sépérateurs* are made for the same purpose as the Harris and Downs segregators, but in these instruments a rubber dam is so arranged that a division is made of the bladder after the instrument has been introduced. They certainly are ingenious but, to my mind, even of less use than our American surgeons' models. Several hundred cases where these instruments have been used successfully have been reported, and they no doubt have a limited use. They require in their manipulation less experience than does the ureter cystoscope, and in normal bladders are fairly reliable. Of course, in using them, if we do not also use the cystoscope, we lose a most important part of the examination, to wit, a view of the bladder and ureteral openings. It can also readily be seen, if the segregator or separator is alone used, unsuspected bladder disease, such as new growths, ulcerations and other abnormal conditions may be overlooked and hemorrhage from them wrongly attributed to some disease of the kidney or ureter.

The late Max Nitze, of Berlin, Germany, in 1887 brought before the medical world the electric cystoscope. For many years before that time, other workers in the same field had been experimenting with methods to view the interior of the bladder. Nitze and Leiter were, however, the first to devise a cystoscope of practical value. It is not necessary in this paper to go into the history of cystoscopy, its great advances and

constant improvements, as shown from year to year in new models. I shall describe here only such parts of this history as seem necessary to make the subject clear.

In the first place, I shall consider the prismatic or indirect vision-cystoscope, which is on the principles of the one devised by Nitze and Leiter; and then the direct vision instrument, the one originally brought before the medical world by Brenner. In both of these instruments we have to deal with systems of lenses and different kinds of lamps for lighting the bladder. Mr. Reinhold Wappler has done much in this country to advance the art of cystoscopy by his mechanical skill and great ingenuity.

In the catheterizing cystoscope of Nitze and modifications like the Bierhoff, Caspar, Albarran, etc., the catheter-chambers are usually placed in the sheath of the instrument. In these instruments the lens system and the sheath are all one piece. In order to change the angle of the catheters as they leave the distal end of the instrument, small tongues are provided which are raised and lowered by a screw at the proximal end of the cystoscope close to the point where the catheters enter the separate chambers. Nitze, Bierhoff and others have devised instruments to be used for intravesical operations. These are more difficult to use than those devised with the straight-vision cystoscopes.

The catheterizing attachment for the direct-view cystoscope, as devised by F. Tilden Brown, is placed on the telescope which, in turn, is separate from the sheath into which it fits. In the original Brenner cystoscope one catheter chamber only was placed on the sheath; in the Brown instrument, two are placed on the telescope. The principle of the outer and inner tubes as used originally by Brenner and later by Brown, enables us to change telescopes without removing the sheath; a great advantage in many ways.

Brown's instrument is a double-barrel, composite irrigating cystoscope of a 24 French calibre. The sheath is oval and about seven inches in length. At the distal end of the sheath is the attachment for the electric current, and on each side are two small openings for the entrance and exit of air or fluid. These are capped with stopcocks. A button-screw is provided on the under surface opposite the point of attachment for the electric current. This screw is used to fasten the telescope

securely in place. Three telescopes may be used at different times in this same sheath: One with a large field for observation, another to carry the catheter chambers on its under surface with a consequently smaller field, and a third for a right angle or prismatic view. In addition, we may use a fourth telescope for retrograde vision. At the proximal ends of the catheterizing telescope are two openings for catheters of No. 5 French, and these are covered with rubber perforated caps to fit snugly to the surface of the catheters to prevent leakage of fluid or air from the bladder. This instrument is now made with an inverted lamp in the beak, so that more light may be brought close to the telescope as it appears at the distal end of the sheath. This light is now so arranged as to render it unnecessary to project the telescope beyond the heel of the sheath. This makes the manipulation of the instrument more comfortable for the patient than it was formerly when the telescope projected for half an inch beyond the sheath into the bladder. The instrument is introduced with an obdurator in place, which is then withdrawn, leaving the sheath in the urethra and bladder. Any telescope, as required, may then be passed down the sheath and screwed in place.

In order to make the accurate and easy withdrawal of the instrument possible, at the same time leaving the catheters in the ureters for a longer time, I devised a new model of Brown's modification of the Brenner cystoscope. I removed the two separate catheter-chambers completely from the catheterizing telescope. In place of them I substituted a short single chamber or bridge at the end of the telescope to support the catheters as they emerge from the sheath. With this model, after the catheters are in the ureters, the telescope is slightly withdrawn to permit the fingers of the left hand to grasp them firmly. The telescope is then slowly withdrawn by the right hand until the short single chamber or bridge is reached. The telescope is then completely removed from the sheath and the catheters lie loose in its lumen. We next advance the catheters gently down its lumen as we remove the sheath. As the heel of the sheath comes to the perineum, pressure there keeps the catheters from being displaced. The catheters then lie in the ureters with the instrument completely out—a matter of considerable importance in a nervous patient or in one in whom the pathological condition present is causing pain.

In addition to this advantage, my model permits the use of small instruments, forceps, curette, etc., for bladder work of a surgical nature. By the use of these forceps, foreign bodies may be removed from the bladder; small stones from the ureteral opening dislodged; portions of new growths obtained for examination, and applications made to ulcers with increased dexterity. No doubt this field will be much widened. My model does away with the two long, closed chambers difficult to properly and quickly disinfect. The light arrangement and sheath are the same in both these cystoscopes, the difference lying in the construction of the catheter arrangement.

Bransford Lewis has devised many ingenious instruments for intravesical manipulation. All these instruments may be used with air or fluid dilatation, although, personally I prefer to employ fluid, having found it less uncomfortable to the patient.

In addition to the cystoscopes mentioned above, we have the open-tube cystoscope, as devised by Pawlik and later introduced into this country by Kelly. This instrument has been found very useful in the female for observation and in the knee-chest position for ureter catheterization. The bladder, by gravitation, permits dilatation with air, and a very excellent unmagnified view may be obtained and small intravesical operations performed.

The open-tube method has also been used in the male in a few experimental cases by Kelly and more recently by Luys in Paris. The latter has devised a special instrument for the purpose, and he has written a book on the subject. Before this work of Kelly and Luys was known to me, I had made various attempts to examine the male bladder by the use of the open tube. Various postures had been tried by me, and recently I have had constructed a special open-tube cystoscope with inverted light for use in the male. I call it a bladder speculum, and I feel convinced that before long we shall be doing much more for diseases of the male bladder by the open short tube. It certainly is practical and requires only persistence to develop it.

The female bladder can be readily cystoscoped and the ureters catheterized with either the direct or indirect view instruments already described. They need not be so long, but it is of no particular inconvenience to have them the same length as the cystoscopes used in the male.

The particular advantages of the open air tube method in the male are the facility with which we can approach ulcers, small

growths, and also the fact that in not using any magnifying power we obtain a perfectly natural picture of the bladder as it really is.

With the direct-view instrument we can, as a rule, see the two-thirds of the bladder further from the outlet better, while with the prismatic type instrument we can usually see better the two-thirds of the bladder nearest the bladder neck. This rule, of course, is an arbitrary one, and we find many variations in it. We should, however, be able to use both types of instrument with equal facility. In spite, however, of our honest efforts to do this, each individual physician will have leanings towards one or the other cystoscopes for most of his work. For many years after Brenner brought out his direct view instrument in Germany, the method was generally condemned and finally abandoned. The main reasons for this were owing to the right-angle position of this instrument's beak which caused much pain to the patient on its introduction, and because of the one catheter it carried being brought out above the field of vision. This made it well nigh impossible to catheterize a ureter, and it was changed later on. With Tilden Brown's type of cystoscope, which has a correct curve to the beak, the instrument is easily introduced and catheterization made comparatively easy. With other models following the original Brenner instrument, the method has become very generally the one of choice in this country, and some European surgeons now employ it with satisfaction in their work.

FRESH AIR WORK FOR SURGICAL TUBERCULOSIS.

BY HENRY LING TAYLOR, M.D.
Professor of Orthopedic Surgery, New York Post-Graduate Medical School and Hospital.

The crowning achievement of the anti-tuberculosis crusade is to have brought medical art into touch with the life of the common people. This paper is intended to illustrate this point of view.

Modern therapy has emphasized the malign influence of contagion, debility and evil surroundings, but it has also shown that even a depraved constitution will readily respond to a favorable environment. It has therefore scotched once for all the ancient bugaboo of inheritance. The battle between depressing and uplifting forces is being waged all along the line, and gradually fresh air is being brought into the hospital, home and daily life, and the patient is being brought out into the air.

Surgical tuberculosis is the same disease as medical tuberculosis, and the general indication for invigoration is the same. Our patient, no matter where the disease is located, must be fortified in his battle with invading microbes, by fresh, cool air, sunlight, a nourishing diet, rest, and a cheerful and tranquil mind. In addition, the joint case will need local rest and protection enforced by fixation and traction splints, and sometimes surgical interference. We thus come back by another route very close to the idea of ambulatory treatment of the American orthopedic pioneers, who devised apparatus to enforce local rest without confinement of the whole body expressly for the purpose of giving their patients the benefit of fresh air.

While under mechanical and surgical care in the hospital, the patients should be provided with hoods, wrappers, comforters and screens, and the windows should be kept continuously open. The nurses should wear sweaters, and the ward temperature in the cool months should not rise above 60° in the day time, and 50° at night; they should preferably be much colder. Cots should be placed on the roof in cold weather as well as in mild. None of these ideals has as yet been realized in our own orthopedic ward.

The Southampton Summer Home, generously supported by

a circle of ladies among the summer residents, takes some twenty children with tuberculous joints from the Post-Graduate Hospital and gives them continuous fresh-air treatment, with excellent care, and a generous diet, from May to November. The home is in charge of two nurses trained in the care of joint tuberculosis, including the application of dressings and splints. The Home is visited once a month by the surgeon in charge. From November to May the same nurses carry out the fresh air treatment in the city tenement homes of the patients, as is soon to be described.

The patients are carefully selected from the convalescents who need invigoration, and the mechanical and surgical treatment of the children taken is carefully carried out. Cases requiring constant surgical attention are not taken, it having been found that such do much better in the hospital, until convalescent.

During the first month at Southampton, the children seem languid, tire easily, and gain little weight; afterwards they become brighter, the appetite and color improve, and considerable gains in weight are made. Experience has shown that a stay of several months is necessary in order to get decided and lasting benefit. Possibly more could be done in a large country or seaside hospital open all the year with a resident surgeon, yet the results obtained in the three years the Home has been open to our patients are positive and even surprising. It should be stated that the home is half a mile from the shore.

Here are the statistics in brief.

	1905	1906	1907
Months open	4½	6	6
Number of joint cases remaining over 1 month.	15	19	19
Number who gained in weight	15	19	19
Average gain in pounds	5.9	7	8
Russell's Emulsion given	yes	no	no
Number of sinus cases	11	11	8
Average gain sinus cases	5.7	8	9
Number of sinuses	30	27	22
Healed during season	7	11	12

One case of osteomyelitis with suppuration is included in the statistics for 1906, and two in 1907; all the rest were tuberculous.

It will be noticed that all the children gained in weight, and that in 1906 and 1907, those who had sinuses gained more than those who had none. The gains in weight and otherwise were

inversely proportional to the extent and activity of the pathological process. Spinal cases gained somewhat less than others.

The statistics convey an inadequate idea of the substantial gain in health and vigor of nearly all the children who go to Southampton. Dr. Ogilvy has shown that the opsonic index in these children is considerably higher than in similar cases in the city. Numbers of children were entirely cured of their joint disease during the summer, and were able to leave off their braces. These were, it is true, either cured cases who were still wearing splints as a precaution, or children who were nearly well when sent, but it is fair to say that convalescence was hastened. It should also be remembered that such results are regularly obtained in a large percentage of cases under mechanical treatment without the advantage of summer outings, and special fresh air therapy.

Yet with all reservations the gain in vigor is marked and important, and when the children return to their tenement homes at the end of the season, it becomes necessary to safeguard this gain already acquired. Even if an all-the-year-round hospital for convalescents and chronic cases were established, the disadvantages of separating a child from its home life for long periods would be keenly felt, for it is probable that even a very poor home is in important respects better, except for short periods, than the most excellent institution. It was felt that the fresh air work must be carried into the tenements themselves as an aid both to the work of the summer home and that of the hospital and clinic.

To meet this need the conditions in the New York tenement houses of our patients were studied; a fund was contributed by friends of the orthopedic ward of the Post-Graduate Hospital and of the Southampton Summer Home, and the experienced and interested matron of the Summer Home was engaged as a visiting nurse to do for our tuberculous joints in the tenements just what should be and has been done for pulmonary cases that cannot be sent away; and also to attend to necessary dressings, and keep the patients in touch with the surgeon, clinic and hospital. The following is the method of procedure. When a case of joint tuberculosis visits the clinic or is discharged from the hospital or summer home, the nurse visits the family, inspects the rooms, and makes a sociologic as well as a hygienic study

FIG. 1.—Fresh air on the fire-escape. Case I, tuberculosis of the hip.

FIG. 2.—Fresh air on tenement roof. Case II, tuberculosis of the hip.

(Taylor)

of the conditions. The number of persons in the family, their health, their earnings, the rental and other expenses, the number and condition of the rooms, the ventilation and lighting and the habits and efficiency of the parents are ascertained and noted. The patient is usually found sleeping in the same bed with others in a dark, unventilated inside room. The premises are often dirty and the food improper or insufficient. There is hygienic and probably economic distress due to incompetence, shiftlessness, intemperance, ill health or bad management. The cause of the maladjustment must be diagnosticated and so far as possible removed. The first step is to explain the patient's needs to the parents and enlist their coöperation. To their honor be it said, they have usually made all necessary sacrifices with astonishing cheerfulness. The next step is to decide if the rooms can be utilized for hygienic living. A few examples will illustrate actual conditions, and the details of management.

Case I. (Fig. 1), was a boy of 12, living on the upper west side, with tuberculosis of the hip, seven sinuses and waxy viscera. The parents were above the average in intelligence and earning power, though the father was intemperate; there were six delicate children ranging from 1½ to 15 years; the family resources had been drained by repeated illnesses, and the parents were exhausted and discouraged. The windows could not be opened on account of the proximity of the elevated road. The necessity for the fresh-air treatment in the home was explained and, after some hesitation, accepted by the mother in spite of the father's opposition, and the family was moved into a better flat on another street. The difference in rent, a cot with warm bedding and wraps, and a daily supply of milk and eggs were provided from the fund. The front room was commandeered for the invalid and the cot was installed next to the window, which was kept open day and night in all weather. The boy passed most of his time on this cot, but was occasionally moved to the fire escape on a wheeled chair. His sinuses were dressed several times a week. When the writer visited this patient six weeks later, the thermometer indicated 36° F. in the room at noon, and the mother was enthusiastic about the treatment, though it involved the partial banishment of the family from their best room. She stated that not only had the patient improved, but that all the other children had lost their colds and snuffles and had gained in color and vigor. This patient, in spite of his

desperate condition gained 7 lbs. during the winter and two of his sinuses closed. It is interesting to note that the father, who had held aloof, soon became interested and without admonition from us, became more temperate in his habits.

Case II. (Fig. 2).—On the lower east side we found the family of one of our patients, a little girl with hip tuberculosis, living in three rooms, one a mere dark closet. The family consisted of the parents and seven small children ranging from a baby up. In spite of the crowded quarters these people managed to take five men roomers, making fourteen people at night in the three rooms. The trouble here was not poverty but thrift; the parents were making money at the expense of the health of the family. The mother had a small baby and refused to open the window; they also refused to move or to get rid of their roomers, and the situation seemed well nigh hopeless. However, we put a cot on the roof, where our patient passed many hours daily. Soon we began to notice changes in the rooms; a folding bed was substituted for the large double bed in the largest room, which served at once as kitchen, dining-room, parlor and bed-room, and things began to take on a neater and more shipshape appearance; the following year the family moved into four larger and better rooms. This patient has spent her summers at Southampton, and is well on the road to complete recovery.

Case III.—In three dark, close rear rooms in the Ghetto lives a family consisting of the parents and six children, one of whom, at the time I visited them, was in an asylum. Our patient was a little boy of four with tuberculosis of the left knee. As I entered the room I saw and smelt the remains of the last meal upon the table; dirt and disorder reigned supreme. On a cot lay the father, claiming to be sick; he had the year before, had his left leg amputated for knee disease, having made a trip to Vienna for the purpose. As a matter of fact the man is incorrigibly lazy and makes no attempt to work. The oldest daughter was on a couch suffering with grave disease of the heart. Two pale babies were asleep, one on a pillow, the other on a chair. The mother, a cheerful slattern, was gossiping with two women friends. This family is supported by charity, and it is impossible to improve the home conditions. We furnish milk and eggs to the boy and take him to Southampton in the summer. It might be better to remove him from the family entirely, but remarkable to relate, he has done well.

FIG. 3.—Fresh air through the open window. Case VI, tuberculosis of the spine.

FIG. 4.—Fresh air on the porch. Case VIII, tuberculosis of the ankle.
(Taylor)

Fig. 5.—Fresh air in front yard. Case of tuberculosis of the spine.

Fig. 6.—Fresh air in a city park. Case of spinal tuberculosis on a frame.
(Taylor)

Case IV.—Our patient, a little girl of three with a tuberculous ankle, as well as her elder sister of fourteen, had undoubtedly been infected by the mother who had recently died of consumption after a long illness. The sister, whose lungs were affected, took entire charge of the father and five children, including cooking, washing and cleaning; she also went to evening school, and brought her little sister to the dispensary. In spite of damp and dirty rooms and insufficient food, our patient made a good recovery from an operation for the removal of diseased bone, at the Post-Graduate Hospital, and is now practically well. We have recently moved this family into better rooms and have hired a woman to come in once a week to do the washing and cleaning.

Case V.—A boy of sixteen, on the upper east side, was in a desperate condition with a tuberculous knee with sinuses. The entire work of the family was done by an invalid sister nineteen years old, whose sight and hearing were seriously impaired. This boy, after an operation at the Post-Graduate Hospital, and fresh air treatment in his home, made a perfect recovery.

Case VI (Fig. 3).—A boy of six with tuberculosis of the spine had been infected by a consumptive father. One of the sisters had had tuberculous glands of the neck, another had had tuberculosis of the hip. The mother was an excellent woman and a neat housekeeper. The boy has done well under the open window treatment with summer outings at Southampton. The two sisters have recovered, and the father's trouble is quiescent.

Case VII.—In the Brooklyn Ghetto, a girl of fourteen with left hip tuberculosis with abscesses, was found with five younger brothers and sisters in three comfortable rooms. The father was a laborer at $2.00 a day; both parents were intelligent, respectable and industrious, and anxious to coöperate. The case is a severe one, but is making good progress after operative treatment, under excellent home care, the open-window and summers at Southampton.

Case VIII (Fig. 4), was a girl of seventeen, who was treated at the Hospital for the Ruptured and Crippled for tuberculosis of the tarsus with multiple sinuses. It was thought that amputation was inevitable, but it was finally decided to try to save the foot. After leaving the hospital, the open window treatment was carried out in the home and the summers were passed in the country; she now has an excellent foot and is the picture

of health. This family consisted of a widow with six children from 9 to 19. The combined earnings amounted to fifteen dollars a week; they had lived for years in the same three-room flat which was as neat as wax. This patient passed considerable time on the porch and in the yard.

Case IX, is one of the most remarkable instances of vicious inheritance that has come under our notice. The patient is a boy of seven with tuberculosis of the spine. The mother, who is living and apparently healthy, states that she lost her father mother, six brothers and five sisters with phthisis, the children dying between the ages of 4 and 22. The husband had three sisters and an aunt insane, and himself disappeared while insane five years ago. Of the six children living the oldest has phthisis, the third has been cured of a tuberculous knee; our patient recovering from a tuberculous spine, is the youngest. The remaining three are said to be healthy.

These are a few examples of the material we have to deal with, and our experience teaches that no matter how bad the inheritance, how depraved the physique, or how hopeless the surroundings, if the case is attacked with a definite plan and a moderate outlay, the results in a large majority of the cases are good, and good in proportion to the thoroughness with which the hygienic treatment is carried out. We utilize, as is shown in the photographs, not only the open window, but roofs, yards, porches, fire escapes, and nearby parks (See illustrations).* We study the family life, strengthening the weak, and utilizing the strong points, and being hopeful and enthusiastic ourselves, we inspire hope and enthusiasm. By maintaining this branch of the work, results have been accomplished that would otherwise have been impossible. Moreover the hospital work itself cannot be conducted intelligently without some knowledge of the home conditions in each case.

My main conclusion is that in addition to the hospital, clinic, and summer home, an orthopedic service should have a visiting department with nurse and physician to organize and carry on the fresh-air work in the home, to keep the patient in closer touch with clinic and hospital, and to coördinate and strengthen all the phases of the work.

* I am indebted for reports and photographs to our visiting nurse, Miss Melvin.

A PLEA FOR THE SUPERIORITY OF PANAS' OPERATION FOR CONVERGENT AND DIVERGENT STRABISMUS. ANALYSIS OF 396 OPERATED PATIENTS.

BY EDWARD S. PECK, M.D.

Professor of Diseases of the Eye, New York Post-Graduate Medical School and Hospital.

This brief contribution on a trite subject in ophthalmic surgery, by a method comparatively new but eminently successful, is based upon sufficient clinical results to entitle it to credence. In two former papers on this topic,* the pathology of strabismus, with a study of the various scientific theories as to its causation and possibilities, was elaborated in some detail. It is essential to the clinical feature† of the present contribution to confine its trend to the behavior of the ocular muscles involved in the strabismic act, convergent and divergent, and to the propriety and limitations of the necessary surgical acts.

The literature of strabismus is voluminous and antedates the scientific literature of ophthalmology itself. Contributions of value from an historic, if not from a surgical point of view, go farther back than contributions to the plastic surgery of the eyelids and adjacent parts of the face. The discovery of the ophthalmoscope by Helmholtz; the treatise by Donders, "Errors of Refraction and Accommodation;" the studies of Javal and the accomplishment of the Javal-Schiotz ophthalmometer; the contributions of Horner, Roosa, Ely and de Schweintz on hyperopia and strabismus; of Horner, Roosa and Weber on errors of refraction, especially astigmatism and their causative association with headache, migraine, conjunctivitis and blepharitis; the studies of Valk, Savage and Stevens on the physiology of ocular muscles and their relative dynamic power with reference to eye-strain and strabismus—all these, and many more of acceptable worth, are contributions to the many-

* The Pathology of Strabismus. The Operation of Panas of Paris. Clinical Analysis of 106 Cases *Medical Brief*, St. Louis, November, 1903.

† A Contribution to the Study of Strabismus, with Especial Reference to the Operation of Panas for its Relief. An Analysis of 225 Operations on 120 Subjects. *Post-Graduate*, July, 1905.

sided study of strabismus, whose correct impetus was initiated and elaborated by Helmholtz's introduction of the ophthalmoscope in 1851. Not until this most important scientific addition to the study of the eye was there believed to be a connection between the anatomical depth and shallowness of the eyeball or between certain intrinsic diseases of the eye and refractive anomalies, now regarded as established facts; more than this, the importance of the angle α (alpha), of the depth of the *fovea centralis*, of the excess of rods and the absence of cones and of the large number of nerve cells and nerve fibres at the fovea, of the coördinate axes of vision, of disturbances in the cornea, lens and vitreous body, and lastly of the relative dynamic disparity between the ocular muscles themselves—I say, the importance of each and all of these points plays a rôle in the study of strabismus, unrecognized previously to the introduction of the ophthalmoscope. Their knowledge is not only interesting, but important. In the light of successful achievement of a surgical act, however, their importance is secondary.

The pertinent question, asked by this paper, is this: Shall one, about to undertake the care and responsibility of a case of established strabismus, with its accompaniment of limited vision, misplaced and distorted planes of vision, circumscribed and falsely projected fields of vision, loss of parallelism of axes of vision, and consequently loss of fixation point, inequalities of right and left vision, gradually increasing spaticity of the internal recti in convergent squint, gradually increasing deterioration of the internal recti in divergent squint, with the presence of headache, eye-strain of manifold character, stooping shoulders, and physical impairment of varied form—shall one allow his judgment to be primarily influenced by physiological and functional facts to such extent that, a simpler and less painful form of operation shall be selected, because, on the one hand, its simplicity and comparative lack of pains, and on the other hand, its possibility of completion at a later date, stand ever before him?

Granted, that a surgical interference for the cure of strabismus has been determined upon, is it not better to complete the surgical act in one session on both eyes, as the Panas' operation demands? It is in the end a saving of pain, a saving of time, and is attended with less expense—all factors of weight in every

social class where strabismus offends. This applies *par excellence* in the lives of school children, who form the very large majority of these patients. From the fifth to the twentieth years of life we find strabismus to predominate; while from the second to the seventh year of life it originates from various causes. To my mind, the argument admits of no question as to the proper procedure—and I am not speaking of only these cases in which surgical interference is obviously indicated, and offers the only hope of attainment of cure. Cases of functional squint, properly amenable to orthoptic and educational treatment, are excluded. Far be it from the arguments of this paper to contend that all cases of squint must, because such, be operated on. All students in this department know that from 25 to 33 per cent. of cases of initial and functional strabismus can be successfully treated by judicious orthoptic measures.

Careful examinations have determined that 80% of the new-born are hyperopic; of these 80%, 60% have strabismus in greater or less degree; and, depending on the age of development of the strabismus, and its determining cause, a very small per cent. are self-curable. The self-adjusting and self-adjustable cases occur before the eighth year of life; rarely does one see such an ideal physiological result, without any interference, as late as the tenth year of life. The unfortunate factor of the let-alone method; or, must I say it? the *nole metangere*, neglect is dangerous delay, whether hope proves more dominant than judgment.

A brief description of Panas' method of operation is as follows:

I offer a liberal translation of his own communication to the Academy of Medicine, Paris, session of July, 1898. It was his second paper on this subject, and is published in full in the *Archives d'Ophtalmologie*, July, 1898, p. 401. The patient being chloroformed, the usual antiseptic means are employed, by preference a solution of biniodid of mercury, so universally used in Paris hospitals. The conjunctival fold is seized horizontally over the rectus tendon, and tendon and subconjunctival fascia are incised. A short strabismus hook is introduced deeply into the buttonhole, and by a brisk movement in circumduction, tendon and tendinous envelope are embraced within the hook. Holding the loaded hook up in the air, slowly progressive tractions are made without jerks or twitches, until the inner border

of the cornea attains without much resistance the external lid commisure. Panas never succeeded in making the least rupture of the tendon of the muscle under traction—an accident not likely to be made when one recalls that, with a weight of five kilogrammes (about 10 lbs.), the muscle of dogs could not be broken. Spasmodic jerks must not be made, as a feeble muscle might be broken. Having completed the elongation of the muscle, one proceeds in the usual way to a complete tenotomy of the tendon down to the surface of the sclerotic; to be assured that no adhesion or tendon remains, the hook is passed to the upper and lower border of the muscle, and every remnant of tendon or attachment to the sclera is cut free. The hook is then released, bleeding checked, and the conjunctival wound is closed by a suture of catgut, and the conjunctival sac irrigated. Precisely the same procedures are carried out immediately on the other eye.

At first, under fear of an over-correction, especially when the deviation was mild, not exceeding 10° to 15°, traction was limited to the eye with fixed strabismus, while a simple tenotomy was made on the other eye; but clinical experience gave overwhelming proof to the author of the operation that elongation of the muscle in each eye was a condition of success; and since then he has always executed this double procedure. Both eyes must be kept bandaged 24 to 48 hours. Aside from a small amount of ecchymosis at the point of traction and tenotomy, conjunctiva and sclera preserve their normal aspect; in five or six days, when the suture either falls out, or is absorbed, cicatrization is perfect. After a proper correction of the ametropic and astigmatic errors, usually present in such cases, the patient is discharged about the 10th day. In order to perfect the establishment of binocular vision, exercises by the stereoscope or other orthoptic means should be begun, and continued a long time with intelligence and persistency. Such is the description given by Panas of this simple operation. The salient points are two: first, the operation must always be done on the two eyes simultaneously; second, elongation and tenotomy of the same muscle must be complete.

The operation hitherto made under the term of strabotomy, was that of *simple* tenotomy; that is, tenotomy of a single, usually the chiefly deviating muscle, and confined to the main,

or central, tendon of that muscle. Various modifications of this simple tenotomy had been made by Arlt, von Græfe, Critchett, Prince, Schweigger, Schellen, Stevens and others. All are modeled after the tenotomy of Dieffenbach, who first performed muscle-division with a tenotome in 1839. The suggestion came from Stromeyer, and was inspired by his results in myotomy on the cadaver in 1838. Guèrin, at the same time made a subconjunctival myotomy of the *rectus internus* with a sharp-pointed knife, by cutting upwards against the rectus tendon, just as Stromeyer severed the *tendo-Achilles* by a subintegumental incision.

Simple tenotomy has always been accompanied by anxiety as to sufficient correction of the deformity. No operator, however practised or clever he may be, undertakes operative interference for the relief of strabismus without feeling much anxiety as to the results in one session; this anxiety is not on account of over-correction, but on account of under-correction, with a possible necessity of further operative interference. The emphatic caution of certain text-books, not to cut too much, has never appealed to me as wise teaching.

In a series of about 3400 cases, I have seen the result of over-correction but 15 times, and these cases were observed from one to twenty years after operation. Of these 15 over-corrections, six required advancement of the over-tenotomized muscle, or tenotomy of the antagonist, while the remainder acquired parallelism by the stereoscope and atropine or by suitable refractive glasses, fortified by prisms. I think it may be stated, with strict adherence to truth, that not more than 40% of strabismics operated on by simple tenotomy of one eye acquire parallelism for life. Some authors place the figures of success lower than this. Serious objection, from a scientific and surgical point of view, as also from consideration of the personal equation of the patient, may properly be made to any method which involves the necessity of repetition and in many instances multiple repetition. The so-called "dosage of tenotomy" is itself a malady to be cured. None of these methods of simple tenotomy offers in advance any definite guarantee of full, complete success in one session.

In the earlier operations of Professor Panas, the deviating muscle of each eye was stretched once. I have found this

inadequate to success in the older cases; and in the higher grades of refractive anomaly. That is to say, given a long lapse of years of strabismus and a fixed, or positive, strabismus, one traction followed by tenotomy has not always been followed by success in one session. Then, too, strong large interni in adolescent subjects with high hyperopia require more than one traction. Strabismics of five and six years of age with 10° to 25° of convergence do not require the surgical interference necessary in cases of 25 to 35 years of age, whose occupations take them in a varied environment. As a rule, women and girls do not require as much traction as men and boys.

Experience has led me to the following rules. In strabismics of 4 to 10 years of age, with an hyperopia not exceeding 3 D., where the angle of strabismus is the same, or nearly the same in each eye, my rule is to make two tractions of each *rectus internus* and follow with a complete tenotomy, involving muscles and supra-corneal and infra-corneal processes. In strabismus of 10 to 20 years of age, with an hyperopia not exceeding 5 D., my rule is to make three tractions of each internus muscle and follow with a complete tenotomy of muscle and all accessory fibres.

In strabismics of 20 to 30 years of age, with an hyperopia of 5 to 8 D. four and five tractions are often required on the greater deviating muscle, and those on the less deviating muscle. These are the cases which must be studied with care. Cases of strabismus of 30 to 40 and 45 years of age, with a low degree of hyperopia, require four, five and sometimes six tractions with tenotomy of the greater deviating internus, while three or four tractions with tenotomy are required for the less deviating eye. These cases are often associated with choroiditis, atrophic retinitis, or a disparity of blood-supply between the chorio-capillaris and the vena vorticosa of the choroid, or some other anatomical or pathological anomaly in the background of the eye; or, on the other hand, with spastic contraction of the interni and relative shortening of the belly of the muscle.

It is well known that few strabismics squint equally with each eye; on the contrary, convergence is usually much more highly marked in one eye than in the other—this may occur in the early development of squint, in the earliest years of life.

It is equally well known that in divergent strabismus, associated with myopia, while the interni are long and weak, as

compared with those of an emmetropic eye, the externi, on the other hand, do not acquire normal dynamic power.

Surgically, however, every case must be carefully studied as to its age, length of the strabismic act, degree of refraction, amount of astigmatism, angle of strabismus, and angle of diplopia; and, objectively as to the vocation of the strabismic, the involvement of syphilis, or other cachexia, the presence of extrinsic or intrinsic eye-disease, etc., etc.

A CLINICAL STUDY OF FIFTY CASES OF MUSCULAR DYSTROPHY.

Idiopathic Muscular Atrophy, Myopathy.

BY JOSEPH COLLINS, M.D.

Professor of Nervous and Mental Diseases at the New York Post-Graduate Medical School and Hospital.

AND

H. CLIMENKO, M.D.

Instructor in the Department of Nervous and Mental Diseases at the New York Post-Graduate Medical School and Hospital.

General Considerations.—There is a form of muscular disease or "myopathy", attended in the main by wasting, that is called dystrophy. The essential disease proper is supposed to be of the muscle itself, but it is as yet undetermined what constituent of the muscle is first attacked. The disease-process finally involves them all, and before its activity ceases all the components of the muscles disappear. It is to be distinguished from progressive muscular atrophy of spinal origin, that is atrophy the result of disease of the cells of the anterior horns of the spinal cord; from atrophy of neural origin, that is from disease of the motor nerves, and from atrophy of myositic origin by careful study and observation which will usually show that muscular dystrophy has the following more or less constant and characteristic features:

1. Onset in early life. (Infantile and juvenile types to be considered later.)

2. Its occurrence in more than one member of a family, and occasionally as a direct inheritance.

3. The involvement in some instances of a particular portion of the body (Facio-scapulo-humeral and distal types to be considered later), and in other instances the manifestations of the disease in muscles that do not correspond to the distribution of a single motor nerve or functionally associated motor nerves, or to the distribution of motor nerves from a spinal segment.

4. The *absence* clinically of the following phenomena which occur in other forms of progressive muscular atrophy: (*a*)

Fibrillary twitching (spinal form), disturbance of sensation (neural form), qualitative electrical change (all forms), and of involvement of the vegetative functions, swallowing, breathing, evacuating, etc.

5. The brevity of its duration compared with other forms (average duration from 10 to 15 years) and its uninterrupted progress.

The literature of the subject has become enormous.

CLASSIFICATION

The history of the recognition of the dystrophies is an interesting chapter in medicine, and we shall refer to it in some detail later. Unfortunately, there has resulted from the many descriptions of dystrophy, its clinical variation and the attempt to separate several types of the disease, a great confusion of the whole subject. In medical societies there is often to be heard earnest debate and discussion whether a given case belongs to this type or that type of the dystrophics—quite as if it made any difference to which clinical type it conformed, so long as the diagnosis of primary myopathy could be made. Although there is ample justification, from a clinical standpoint, for the division of myopathies into types, *first*, in reference to the age of the individual when it develops (such as (*a*) in infancy, (*b*) in youth), *i.e.* the infantile type and the juvenile type (Erb); *second*, into the part of the body that is first and predominantly affected, such as, (*a*) the face, shoulder and upper arm—facio-scapulo-humeral type, Landouzy-Dejerine—(*b*) the distal type, *i.e.*, the type in which the atrophy begins in the distal parts of the body and remains confined to them for a long time, indeed, for many years before any of the proximal parts of the body are affected); *third*, according to the most permanent objective feature, such as enlargement or apparent hypertrophy of parts of the body, *i.e.* the socalled pseudohypertrophic type (Duchenne's " Pseudohypertrophic muscular paralysis "). No objection can be put forward to such attempt at classification of the dystrophies, providing it be granted that it is done merely to facilitate recognition of the disease clinically, and that such classification is in no way an attempt to separate one form of the disease from another; but it is because of such attempt that confusion has arisen concerning the dystrophies. There have been so many socalled atypical cases published, *i.e.*,

cases that do not absolutely conform to the varieties of the disease that have been enumerated above, that it has seemed to many far better to disregard entirely such attempts at classification; but, in reality, after a sufficiently large number of socalled " atypical " cases shall have been recorded, " types " of the disease will become more definitely established.

There are examples of disease, in which myopathy is apparently the most conspicuous disorder, which have to be classified as allied to the dystrophies. Such cases seem to be most frequently associated with epilepsy and with certain forms of imbecility. We have to admit that they are allied to primary myopathy. In this respect we are in a position analogous to that of the alienist who, though recognizing a fairly typical manic-depressive insanity or dementia-precox, has to classify many cases, perhaps the majority, which he has attempted to put under one or the other of these headings as " allied cases," *i.e.*, allied to manic-depressive insanity or to dementia precox. It may be that eventually we shall classify the dystrophies in some other way than clinically, just as we have been able to classify diseases of the kidneys from the standpoint of the constituent that is first and predominantly affected. In other words, it is quite possible that different forms of dystrophy begin in different constituents of the muscle, but some new method of investigating the muscles must first be discovered before this can come about.

The socalled Neural form of Dystrophy. There is a form of muscular atrophy which is usually considered in the books in conjunction with the dystrophies, which is known as the neural or peroneal type of progressive muscular atrophy. It was first described by Charcot and Marie and afterward more elaborately by Tooth and by Hoffman (known as the Charcot-Marie-Tooth type). The disease is to be distinguished clinically by symptoms which do not occur as a rule in the true dystrophies. In the first place, the atrophy is confined very largely to the peroneal muscles; there are sometimes sensory disturbances, particularly pain; vaso-motor disturbances in the atrophied parts; reaction of degeneration and fibrillary contractions in the atrophying muscles. A few cases of this type have been investigated anatomically, and especially by Marinesco, Sainton, Dejerine, Armand-Delille and Siemerling. In all of these, wide-

spread changes have been found in the spinal cord and in the peripheral motor nerves, and more particularly sclerosis of the posterior columns of the cord; slight degeneration of the pyramidal tracts and columns of Clark; some alteration of the cells of the anterior horns, and more or less degenerative changes in the peripheral nerves or intra-muscular nerve fibres. Such findings show how far removed this disease is from true myopathy, in which there is no disorder whatsoever of the central or peripheral nervous system that can in any way be construed as having a causal relation to the disease of the muscles.

The disease *muscular dystrophy*, or primary myopathy, is the most typical example of abiotrophy. A few years ago Gowers suggested the name " abiotrophy " to indicate a defective vital endurance. Such defective vital endurance, inherent in the embryonal tissue from which the muscular structures of the body arise, constitutes muscular dystrophy or what would be more felicitiously called primary myopathy, as the word dystrophy, itself, means defective nutrition.

We have taken from our records, the histories of fifty successive cases of dystrophy and subjected them to analysis, particularly to learn how they group themselves in regard to the classification that has been mentioned above, and more particularly the pseudohypertrophic type, the juvenile type, the facio-scapulo-humeral type and the distal type, to determine anything of interest in that element without which the disease never apparently develops, viz., defective heredity and, finally, to study the clinical course of the disease, *i.e.*, the disease as a whole and not any special varieties of it. We preface this analysis with a brief review of the early history of the subject, and append some remarks on the morbid anatomy of the disease.

History.—In 1829 Abercrombie described a case of muscular wasting in a boy in whom the musculature of the shoulders and upper arms had almost completely disappeared, while those of the forearms remained intact. Abercrombie was unable to attribute a cause for the affection, but he suggested that the disease was dependent upon changes in the peripheral nerves. About the same time Charles Bell gave an excellent description of muscular atrophy, to which later the name of pseudohypertrophic muscular paralysis was given. The description is to

be found in his book on the nervous system, published in 1830. In 1832, Cruvelier reported a case of general muscular wasting, which he thought was due to destruction of the anterior horn cells, but microscopical examination failed to confirm this opinion. This and a similar case suggested to him that he was dealing with an idiopathic muscular disease. In 1836, Koster and Giaza related the histories of patients who were undoubtedly afflicted with this disease, now known as progressive muscular dystrophy. Cases were also reported by Romberg, in 1846, and Duchenne, in 1847. In 1852, Meryon was the first to report a series of cases in English literature, and was also the first to make a post-mortem examination. To him, more than to anyone else, is due the credit of pointing out that we were not dealing with an amyotrophy (spinal cord atrophy), but with a myopathy. In 1854, Oppenheim read his thesis on: "Lipomatous Degeneration of Muscles", but it was left to the French school, and particularly to Cruvelier, Duchenne and Aran, to show that the various forms of muscular wastings constitute a disease *sui generis*. In 1861, Duchenne first called attention to the fact that in some instances, at least, the volume of the muscles was increased. In the same work Duchenne described a progressive muscular atrophy of children (Arthropathie musculaire progressive de l'enfants) characterized by affection of the facial musculature, as well as those of the limbs and trunk. He likewise pointed out that frequently more than one member of the same family are affected. This disease was again described by Landouzy and Dejerine, in 1885, and it is now commonly associated with their names. They were instrumental in showing that the disease in question is independent of any changes in the nervous system. In 1873, Friedreich reported a series of cases considered to be chronic progressive myositis. He gave the soundest possible reasons for separating these muscular affections from the amyotrophies. In 1875, Leyden proposed to separate the hereditary forms of muscular atrophy from those of the Aran-Duchenne type, *i.e.*, from progressive muscular atrophy of spinal origin, In 1883, Erb combined the various forms of dystrophy under the heading of "Juvenile Muscular Dystrophies" and considered them in contradistinction to atrophies due to changes in the central nervous system, and suggested that the name of muscular dystrophy (meaning difficult nutri-

tion) be given to them. The various types of progressive dystrophies were thus first grouped together by Erb. He also assigned to them heredity as a cause. In 1902, Gowers recorded a case of "distal myopathy," *i.e.*, atrophy of the muscles at the distal ends of the body. Spiller has reported another.

Etiology.—Muscular dystrophies are an expression of prenatal inadequate endowment. The inadequacy may hark back many generations, so many that we cannot trace it, or if we can, it is so unlike the deviation before us that we do not recognize it. Jendrassik thinks that heredity does not play any specific cause, for any part of the muscular apparatus, nervous system, skeletal or connective-tissues may suffer from mal-development, all forms of aplasia atrophies may develop. So far as progressive muscular dystrophy is concerned, a number of observers (Barsikow, Sveaze, Friedreich, Marie and Guion, Bielschowsky, Landouzy-Dejerine, etc.), have reported cases in which direct heredity through the mother was observed. It is obvious that paternal heredity would be lacking, because the male is prevented from procreating by the impotency that comes with the disease contemporaneously with puberty.

The most striking feature of the disease from the point of view of its causation, is its occurrence in more than one member of the same family. Of our fifty cases, twenty showed distinct hereditary taints; in nine instances, two sisters and two brothers were affected; in one instance, three brothers suffered from the disease. In the latter case the father was asthmatic, alcoholic, and had had several attacks of insanity, and a paternal aunt was insane. In another family in which a brother and sister were affected, the parents were first cousins. In ten cases, aunts, uncles or first cousins suffered from dystrophy. In three cases (including the above mentioned case) the father or mother was insane. There were three of our cases whose parents died of tuberculosis, but this disease as well as syphilis does not seem to play an important rôle in the causation. So great a rôle does heredity play in this disease that, in 1884, Friedreich propagated his theory of "Disponierende Diathese," according to which the muscle-tissue during its development acquires a diminished power of resistance and impaired nutritional state, and a gradual disposition to all forms of degenerative diseases.

This theory was revised and elaborated, in 1903, by Gowers in his essay on "Abiotrophy," who considered it the result of defective vital endurance inherent in the embryonic tissue, from which the muscle-structure tissues arise. It has been further supported by Babinski and by Ananof, who have pointed out that those groups of muscles which develop simultaneously in the embryo are also simultaneously affected in dystrophy. This grouping of the affected muscles has also much diagnostic significance.

Mosso considers the atrophy to be a manifestation of universal degeneration. In his 59 cases he found 18 imbeciles, 7 epileptics, 21 skeletal abnormalities, and 22 with trophic disturbances. We are unable to support Mosso's theory. There is, however, one peculiarity worth mentioning in this connection, and that is that in nearly every child with dystrophy, boy or girl, that we have examined, an abnormal growth of hair on the lower extremities has been found.

Age.—The disease is usually said to begin during the latter part of childhood. In our cases the ages varied as follows:

1 at birth, 9 at three years, 6 at five years, 8 at six years, 7 at seven years, 10 at eight years, 5 at ten years, 2 at thirteen years, 1 at seventeen years, 1 at thirty-two years.

From this it is seen that the majority are attacked between the ages of six and eleven years—a time when the musculature is especially in a state of development, as has been shown by Meek. Only one case so far, reported by Pick, has been observed in which the dystrophy began late in life. His patient was upward of 50 years old.

Sex.—Males suffer more frequently from this disease than females. In our series, there were forty males and ten females.

OCCURRENCES PRECEDING THE ONSET OF THE MYOPATHY.

a. *Acute Infectious Disease.*—In 24 of our cases the disease was preceded by some exanthema, scarlet fever predominating; but this may mean absolutely nothing, for it represents probably the proportion of persons of this age that have the acute exanthemata. In one case, that of an Italian boy eight years of age, the disease followed some acute illness which was accompanied by convulsions and lasted two weeks. Immediately after the febrile stage was over the parents noticed that the child was weak and "staggered." This case was at first mistaken for

anterior poliomyelitis, but proved in the course of time to be a typical muscular dystrophy of the pseudohypertrophic type. No doubt, infectious disease may serve as an exciting cause in dystrophy, *i.e.*, it may bring the disease out sooner than would otherwise, have been the case, but it can have nothing to do with causing it.

b. *Trauma.*—Cramer reports the case of a laborer, 35 years of age, who was hit by the hoof of a horse. This was followed by weakness in the affected forearm, and in the course of time developed into a typical muscular dystrophy. In our series, there were two patients in whom it is said that the disease began rather abruptly and developed rapidly, in which a distinct history of traumatism was given; but it is difficult to conceive how trauma can have anything to do with initiating the disease. It may expedite the course and development of it, but it is wholly inadequate to cause it.

PATHOLOGICAL ANATOMY.

The nervous system, central and peripheral, is, as a rule, found intact in the vast majority of cases that have been studied anatomically, but it is quite possible that the lesion may stand in definite relation to disease of the muscle spindles, which forms a striking feature in transverse section of the muscle. It would be an important addition to our knowledge of these elements, and to our knowledge of the myopathies if their condition, if their state of preservation were known in every case of dystrophy. If they are unaffected in every case of dystrophy, then it would seem most likely that the disease must depend upon conditions outside the muscle, and at a place where the motor and sensory fibres are separated *i.e.*, central to the posterior root ganglion. In a lecture on myopathy, delivered six years ago, Gowers rejects rather disdainfully, not to say scornfully, the idea that myopathy may be a trophoneurosis. "It is a doctrine that should have died of inanition long ago; purely hypothetical, every definite fact is opposed to it."[1] That, as an expression of personal opinion, should have some weight; but in so far as it scorns the opinions of others and the testimony of post mortem investigations, which show that a condition of the muscles similar to that

1. *British Med. Journ.* July 12, 1902.

found in the myopathies is found also after a spinal lesion, it should be disregarded.

Those who have reported changes fail to agree as to the constancy of these changes. Babes found the motor-end plates undeveloped. Strümpell, and Sachs and Brooks reported a case in which there was shrinkage of the cells in the dorsal ganglia. In our two cases that came to autopsy, no changes in the central nervous system were found. A considerable number of cases have been reported in which there were pathological alterations in the anterior horn cells of the spinal cord[1] consisting in diminution of the number of cells, alteration in the size and shape of the cells, and change in the constitution of the cell bodies. These changes are to be interpreted as secondary to the primary muscle disease. Why they occur in some cases and not in others has not been satisfactorily explained. They are not unlike the changes that occur in the anterior horn cells after amputation of an extremity.

The real lesion is in the muscles themselves. Here one sees hypertrophy and atrophy of the muscle fibres going on hand in hand. Together with this there is increase in the interstitial connective tissue with a large deposit of fat. Much controversy still exists as to whether or not the disease begins primarily in the connective-tissue or in the muscle-fibre proper. Duchenne, Charcot, Sachs and Brooks, Berger, and others found a proliferation of fat and connective-tissue followed subsequently by atrophy of the muscle fibres. Berger excised a piece of calf muscle in a boy suffering from typical symptoms of pseudohypertrophy. Microscopical examination showed an increase of connective-tissue. A few years later, post mortem examination of the same case showed that this connective-tissue had changed into fat. Erb and Lorenz, on the other hand, found degeneration of muscle fibres preceding the proliferation of fat and connective-tissue. This divergence of opinion is apparently due to a variety of changes produced in this disease.

In general, it may be said that as far as the pathological anatomy is concerned, the changes in the muscles may be classified under

1. Erb and Schultze, *Archiv. f. Psychiatrie*, Vol. 9; Kahler, *Zeitschr. f. Heilkunde*, 1884; Strümpell, *Deutsche Zeitschrift f. Nervenheilk*, Vol. III; Kollarits, *Deutsches Archiv. f. klin. Med.*, Vol. LXX; Ingbert, *Journal of Nervous and Mental Disease*. Vol, XXXIV; Holmes, *Review of Neurology and Psychiatry*, Vol. VI, *et al.*

two headings: First, the sclerotic type, in which the connective-tissue predominates; and secondly, the pseudo-hypertrophic type, in which the adipose tissue predominates. Not infrequently both these changes go hand in hand. In the sclerotic form, *i.e.* when connective-tissue predominates, the muscles are of a brown or rose-color and are thin and hard. When far advanced, such muscles present nothing but bands of fibrous tissue. In the pseudo-hypertrophic type, the muscle is of a deep red color with a light yellowish tinge, due to the deposit of fat. Its consistency is soft and flabby. Bands of connective-tissue containing layers and bundles of fat, pass through the substance of the muscle.

Microscopically, besides an increase in connective-tissue and fat cells, proliferation of the nuclei and muscle-fibres is the first change to be observed. This is soon followed by a division and hypertrophy of the fibres themselves. The contour of an individual fibre is rounded, and on cross-section it has acquired a circular outline instead of its normal polygonal. Soon the nuclei disappear; the fibres themselves begin to show various stages of atrophy with vacuolisation in the sarcolemma, so that a typical atrophy of the fibre may be seen next to an hypertrophied fibre. The interstitial fibrous tissue is increased, especially in the localities of hypertrophied muscles. A thickening of the adventitia and the blood-vessels is often observed. The deposit of fat in the interstitial tissues varies in amount. Lorenz subdivides the dystrophies according to this deposit, into atrophies, sclerotic and hypertrophic lipomatosis.

SYMPTOMATOLOGY.

The onset of all varieties of muscular dystrophy is insidious. The first symptom noticed in all forms, except the facio-scapulo-humeral type, is some disturbance of gait. The almost invariable complaint (in 35 of our cases) is that the patient's legs are weak, and that he falls on the slightest excitement. This is most noticeable on some such effort as ascending stairs, which is either difficult or impossible. This is often attributed to the youth of the child, and parents console themselves with the idea that the patient will eventually "outgrow" it. In fact, they do not seem to be at all alarmed about the child's condition, since physically it looks better nourished than other normal children. In five of our cases, the patients had never been able to use their limbs as well as other children

at corresponding ages. The eye of the layman, of course, cannot detect any fault in children who are apparently so well nourished. A little later on, however, another very characteristic symptom appears, *i.e.*, the mode of rising from the recumbent position. In order to assume the erect posture the patient is obliged to undergo a series of peculiar movements which are almost uniform in all cases. (see Fig. No. 1). If lying on the back, the patient first gradually turns over on the abdomen; then, while supporting himself on his hands, he raises the trunk high enough to enable him to get on his knees, the hands still being on the ground; then the knees are raised, and the patient is now in a position in which only the hands and feet touch the ground. Next, he supports his hands on the corresponding knees, gradually he brings the hands upward along the thighs and, finally, with a sudden upward jerk, the body is brought to the erect position. All these movements are very slow, and it often takes five to ten minutes before the patient finally achieves the erect posture. Later on, when the extensors of the back are involved, this mode of rising becomes impossible and the patient is unable to rise at all.

The gait is peculiar; frequently it is waddling in character, resembling that of the duck. The legs are raised with difficulty, often dragged along the ground, and the trunk, including the pelvis, is thrown in the opposite direction, which gives the body an oscillating form of motion. In walking upstairs (see Fig. 2) the patient is obliged to stand on one foot while he slowly brings the other up, and then supporting himself on the knee of the resting foot with one hand, he slowly pulls himself up the stairs with the other. At this stage of the disease the tendon-jerks, such as the knee-jerk and the ankle-jerk, are very much diminished, and later on disappear altogether. It is worth mentioning here, that with diminution or absence of the tendon reflexes, the direct myotatic irritability is also either diminished or absent. This is unlike those conditions in which the absence of the tendon reflexes is dependent upon some break in the reflex arc and not upon disintegration of the muscle. Thus, in locomotor ataxia, in multiple neuritis, (before the atrophic stage) and immediately after death, conditions in which the tendon-reflexes are absent, the direct myotatic irritability is increased. The greatest

FIG. 1.—To illustrate a "critical moment" in assuming the erect posture. He must either "snap into place" or crawl upward with the hands on the legs.

(Collins.)

FIG. 2.—To illustrate the way the myopathic patient goes upstairs.

myotatic irritability in any case observed by us was immediately after death.

Early in the disease, long before any actual paralysis is noticed, the face begins to show characteristic changes. It is usually full, motionless, and featureless. The socalled " facies myopathies " or " facies des sphinx " (of Ballet) is well illustrated by Fig. 7. The facial appearance is probably due to involvement of the frontalis, orbicularis and palpebrarum. During the progress of the disease the patient may lose the ability to whistle and to pout the lips. The atrophy of the orbicularis palpebrarum may lead to incomplete closure of the eyelids and even to lagophthalmus.

In the erect position a lordosis (see Fig. 3) is observed In some cases this is almost the first symptom to be noticed. The pelvis and the head are thrown back, probably because the patient throws the main part of the body weight on his feet, thus relieving the muscles of the back and the glutei. It is characteristic in the lordosis that in the early stages it disappears in the sitting posture. When the disease, however, is far advanced, the lordosis is present also in the sitting posture, and in the last stages, instead of a lordosis, the trunk is telescoped into the pelvis, as is well shown by Fig. No. 4. Soon it becomes difficult for the patient even to walk on a level plane; and it is usually at this stage that the child is brought to the clinic. Often fractures and other injuries caused by falls occur. In one of our patients there was fracture of the thigh, in another fracture of the humerus. Gradually, the power of locomotion is lost entirely and the patient becomes bedridden, his lower extremities being a dead-weight. Coincident with the inability to walk, the patient complains of being unable to raise his arms, and still later to flex his elbows. Changes begin to appear in the musculature of the upper extremities, especially in the lower part of the pectoralis major and latissimus dorsi. It is to be noted as was first pointed out by Gowers, that the supraspinatus is almost the last muscle to become affected. Involvement of the musculature in the upper extremities in a typical case begins to appear usually five or six years after the initial symptoms have developed. Among other symptoms when the musculature of the upper extremities is involved, are the characteristic winged-shape scapulæ seen in illustration (Fig. 5). It is a point almost of differential diagnosis that the musculature of the fingers does not become

involved until the very last stages of the disease. Some of our patients who have become entirely a dead-weight, are still able to use their fingers and move their toes.

The abdomen is protruded, due to the large deposit of fat tissue, but the abdominal muscles are rarely affected. When they are involved, death is caused by difficult breathing.

Electrical examination shows diminution of excitability to both currents, but no reaction of degeneration. Later on, when the musculature is completely wasted, no response to the faradic current is obtained. The calves, thighs and buttocks are, in contradistinction to the upper part of the body, thick, soft and flabby. This is due to the deposit of fat, which begins to take place in these muscles.

Working on the basis of the relation of glucose to muscles, special inquiry as to the sugar metabolism was made in some of our patients. Notwithstanding the fact that large doses of glucose were given to patients in the terminal stages, no sugar was detected in the urine. Some observers (Bender *et al.*) have made further inquiry into the metabolism of these patients, but found no changes except a diminution of kreatinin.

Distal Type.—Gowers has reported two cases in which the disease began distally, *i.e.*, in the fingers and the toes. We have the history of one case, which will be cited later, in which the disease began distally. This, however, was an atypical case and was for a long time thought to be a progressive chronic poliomyelitis, but repeated examinations failed to show any reaction of degeneration and the group of muscles involved was characteristic of dystrophy. Subluxation of the shoulder joint is occasionally seen when the muscles around those joints are involved. The muscles of the neck are attacked late in the disease, and although the patient is no longer able to hold the head erect, he is still able to move it from side to side.

Myotonia Atrophica.—One of the most interesting symptomatic associations of the dystrophies is myotonia. Ordinarily the muscular tonus is diminished in dystrophy, but there have been some cases reported by Pelz[1] and by Hunt[2], Curschmann, Gauff, and others, in which myotonia resembling that of Thomsen's disease was present. Satisfactory interpretation of these cases has yet to be furnished.

1. *Archiv. f. Psychiatrie*, Vol. 42, p. 704.
2. *Proced. of the N. Y. Neurological Society*, November, 1908.

FIG. 3.—To illustrate lordosis and characteristic station in early stages of myopathy. Likewise the " wing " scapula.
(Collins)

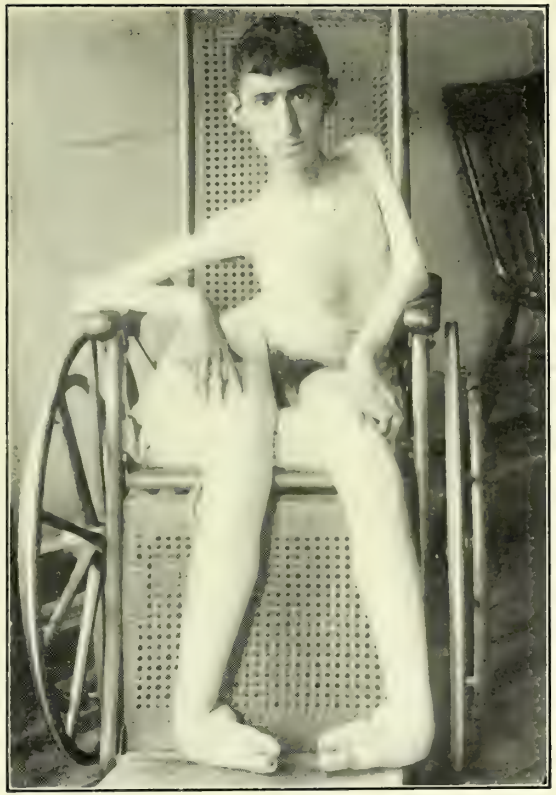

FIG. 4.—To illustrate telescoping of trunk into pelvis and pedal deformities in advanced stages of primary myopathy.

DEFORMITIES ACCOMPANYING THE DYSTROPHIES.

All sorts of reducible deformities make their appearance. There may be complete valgus, varus, equinus, etc., all of which when corrected reappear when left alone (see Fig. 6). There is also ptosis of almost the entire abdominal viscera, due to the deformed back and pelvis. With all this the vegetative organs do not seem to suffer, and the appetite as well as digestion is good. In one of our patients there was often an extraordinary ravenous appetite.

The contractures recurring with dystrophy and the deformities that thereby result have been much discussed and by many writers. Erb maintains that the weight of the hypertrophied muscles is responsible in many cases for these contractures. Hoffa offers the mechanical theory of muscle antagonism. The contractures are present in the non-paralyzed, because the intact muscles are out of function. Friedreich thinks that the contractures are due to the hypertrophy, and later the contracture of interstitial connective-tissue which has a tendency to contract. Landouzy and Dejerine expressed the opinion that the occurrence of contractures was pathognomonic of the infantile type. Raymond, in discussing the deformities occurring in the upper extremities, endeavored to show that the muscles, *i.e.*, the biceps and brachialis anticus appear slightly strained and thus produce contractures at the elbow. This last statement was contradicted by Erb, who saw the contractures in only about 35% of his cases.

Hahn analyzed 67 cases in which the various types of dystrophy are represented. He comes to the following conclusions: Contrary to Landouzy, Dejerine and Raymond, the least number of contractures occur in the infantile type. The greatest number of contractures he finds in the pseudo-hypertrophic form. The most frequent form of deformity is pes equinus and equino-varus, drop-foot, permanent flexion of the large toe. Ankylosis may be a final stage of these contractures. Next to the foot deformities come the permanent flexions of the knee, and then flexions at the hip. Hahn further reports two cases in which the contractures appeared as the first symptom of the disease. He finds that the contractures are an important item in the differential diagnosis.

Contractions often appear in the so-called sclerotic type, and

were noticed in two of our cases. In one they were confined to the Achilles tendon, and in the other to the muscles of the back of the neck, so that the boy walked on his tip-toes holding the head flexed backward.

Schlippe[1] showed that the long bones are atrophied in the thickness of the shaft and not affected in their length, nor are there any changes in the epiphysis and joints of these bones. He, therefore, concludes that this process is not due to an arrest of development, for if such were the case the bone would swell in all its dimensions. On the other hand, it is not due to any trophoneurotic changes. In this instance, as is well known, the bones are unchanged in their thickness, and rarefication of the spongiosa occurs in spots, making the appearance as if the bone were checked. Schlippe further thinks that the skeletal changes are not secondary, but are brought about most probably by the same cause as that productive of the change in the muscles. Dreyer[2] supports this view by the fact that the bone changes are most pronounced where the muscular changes are severe. Schultze[3] reported two cases in sisters. In one there was dystrophy associated with general atrophy of bones, and in the other there was only atrophy of bones without dystrophy. Bregmann[4] records a case of progressive muscular dystrophy in which the bones were markedly hypertrophied. Eulenberg[5] also records a case of progressive muscular dystrophy with hypertrophy of the shafts of the bones in which the epiphyses remained in their normal state. Schultze[6] reports a case of pseudo-hypertrophy with general atrophy of bones. Marquard[7] reports a case of acromegaly associated with progressive muscular dystrophy.

The order in which the muscles are involved may be arranged in the following manner:

Dense, Thickened Muscles.—Calves, sartorius, glutei, triceps, deltoids, infraspinati. *Atrophy.*—Pectoralis major, cucularis, serratus magnus (anterior portion), latissimus dorsi, biceps, quadri-

1. *Deutsche Zeitschr. f. Nervenheilk*, 30.
2. *Deutsche Zeitschrift f. Nervenheilk*, 31.
3. *Deutsche Zeitschr. f. Nervenheilk.* 1899.
4. *Zeitschr. f. Nervenheilkunde*, 1899.
5. *Deutsche med. Wochenschr.* 1896.
6. *Deutsche Zeitschrift f. Nervenheilk.* 1899.
7. *Inaug. Dissert. Berlin*, 1896.

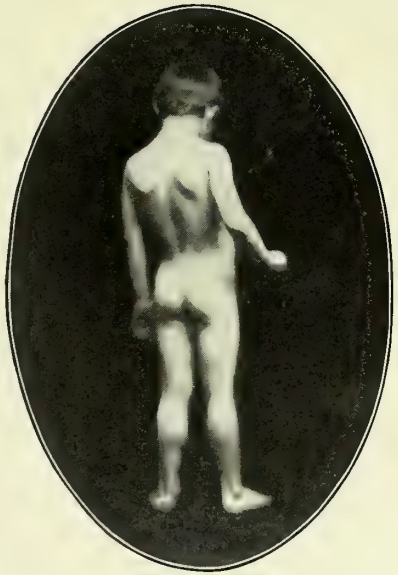

FIG. 5.—Winged scapula in an early stage. Illustrates also typical attitude.

(Collins)

FIG. 6.—"All sorts of reducible deformities make their appearance."

ceps femorus, abductors. Like some other observers, we were unable to determine any interference in muscle function of the voluntary or involuntary cardiac muscles.

DIAGNOSIS.

In well defined cases the disease, when once seen, cannot be mistaken. The age, the facies, the gait, the lordosis, the manner of rising from the recumbent posture, of walking upstairs, are all sufficiently characteristic for a correct diagnosis. In the early stages and in the irregular types, the diagnosis is occasionally difficult. Quite frequently the physician fails to notice the importance of the gravity of the case, and the diagnosis of "weak ankles" is often given to the parents. Yet a careful observation would rarely fail to disclose the real condition. The calves, as a rule, are abnormally large, especially in proportion to the lower extremity. Together with the increase in volume, these muscles are considerably weaker than the others, and the muscular power is very sluggish. Gowers points out a very characteristic early symptom, viz., enlargement of the supraspinatus after wasting of the latissimus dorsi of the lower part and the pectoralis major. The facial expression also is characteristic. Occasionally, however, the volume of the muscles is not changed, and in those cases special attention is to be paid to the function of the muscles in regard to their strength and electrical excitability. It will be found that these two features are far below proportion to the volume. The muscles are weak and the electrical response is feeble.

At times the disease may be mistaken for chronic progressive anterior poliomyelitis, congenital spastic paraplegia, progressive muscular atrophy, syringomyelia, inflammation of the spinal column, and a form of polymyositis described by Oppenheim, Cassirer and Schultze. In chronic anterior poliomyelitis there is present reaction of degeneration, the absence of pseudohypertrophy and the peculiar grouping of the affected musculature which serve as a guide for making the diagnosis. In anterior poliomyelitis the affected muscles correspond to the segment of the cord in which the anterior horn cells are destroyed, while, as pointed out above, in dystrophy they do not follow any segmental arrangement of the cord, but more a so-called embryonic arrangement. Thus we see that

sometimes the muscles of the face and shoulders are affected, in others the calves and back muscles are first affected. Congenital spastic paraplegia is characterized by spasticity and its accompanying syndrome. In syringomyelia, which may occasionally resemble this disease, there is usually dissociation of sensibility, and the small muscles of the hands are, as a rule, first attacked, giving the hand a claw-shaped appearance, the *main en griffe*, and usually show reaction of degeneration. Progressive muscular atrophy begins late in life; it is preceded and accompanied by fibillary twitchings; there is no pseudo-hypertrophy, and the disease usually begins in the small muscles of the hands and travels upward.

TREATMENT.

Nothing can be done to arrest the progress of this disease. The cause for this deficiency in therapy lies evidently in the fact that it is a disease of evolution. As a propyhlactic measure little can be done beyond advising individuals suffering from this disease, having neuropathic tendencies, not to overtax the neuromuscular system. At the same time, however, systematized exercise of the muscles should be practiced. The general care of such individuals, with especial attention to the nutrition, is very important. After the dystrophies have appeared, a great deal can be done in delaying the progress of the disease by proper gymnastics, massage, electricity, diet, and coöperation of the orthopedic surgeon. The type of gymnastics best adapted for this disease is the resistant form. Great care, however, is to be taken that too much strain is not placed upon the atrophied muscles. Gymnastics have benefited especially a few cases of the scapulo-humeral type, and when properly applied they may improve even the pseudo-hypertrophic type.

We consider that electricity, when properly handled, may be of some service. It can prevent, at least, atrophy due to inactivity. When applying electricity to this form of disease, we usually choose the faradic and to a lesser extent the galvanic currents. The former is used for a small number of short contractions of the atrophied muscles without producing any fatigue; while the galvanic, when used moderately, improves the circulation.

Massage, especially the kneading form, tending to increase

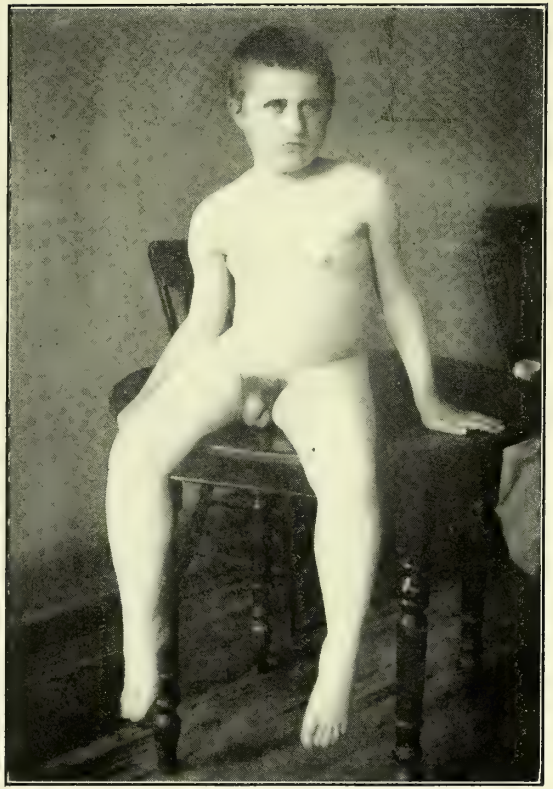

FIG. 7.—Typical facies, attitude and contour of a patient with primary myopathy.
(Collins)

FIG. 9.—To illustrate a typical so-called hypertrophic myopathy of four years' duration.

the metabolism of the muscles, is of small service, and in no way to be compared with systematic exercises.

Great stress should be laid upon rest. The patient is to use as little as possible the affected muscles. The early co-operation with the orthopedic surgeon is most advisable. Contractures and deformities should be corrected as soon as they appear.

We have had no success in any of our cases from the administration of medicines. The thymus and thyroid have both been suggested as curative agents, and although they have received a thorough trial at our hands we have not met with any success. A case has been reported as cured by strychnin; we have not been so fortunate.

ILLUSTRATIVE AND TYPICAL CASES.

As was mentioned above, Gowers reported a case of myopathy or progressive muscular dystrophy, which began in the distal muscles first, *i.e.*, the hands and feet were first attacked. We can report only a single case which appeared to us to be progressive muscular dystrophy of the distal type. The history is as follows:

Distal Type.—E. S., 20 years, single; housework; Russia.

Family History. Parents healthy. Three children died in infancy; one miscarriage (last).

P. H. Patient is the fourth child. Had measles and scarlatina at an early age. Began to menstruate at 17, regular.

P. I. Began 12 years ago. Fell while walking one day. A month later, the mother noticed that the patient limped with the left leg. No pain. The limping was very slight in the beginning, but increased. A physician in Russia could not make out any defect at that time. Five years ago the upper extremities began to tremble and lose strength; first the left, then right. The feet and hands were in the beginning very stout, but became thinner in time. Patient has difficulty in using the hands for fine work, such as sewing, etc.

Status.—Patient of small stature, No Romberg. Gait: Stride long, limps towards left, dragging the left extremity. Innervation of the face: Lines equally indistinct on both sides. Tongue protruded straight, fine tremor, coated; papillæ prominent. Active and passive movements of head unrestricted. Active movements of both shoulders, elbows, and wrists present. All the fingers of left hand at interphalangeal joints can be extended only

to a right angle; further passive extension is possibls. The two inner fingers of the right hand can be extended actively to about 130° at both interphalangeal joints. The gross motor power of the right upper extremity is fairly good; of the left upper extremity diminished in all its three divisions. The muscles of the back and abdomen normal. Difficulty in flexing and extending the left hip and left knee. Gross motor power diminished in the left, and good in the right lower extremity.

Cranial nerves normal.

Viscera: Presystolic murmur heard at 5th interspace, internal to midclavicular line, propagated upwards and to the left. Lungs and abdominal viscera normal.

Atrophies.—Atrophy of the fingers of both hands, the left more than the right (is able to button and unbutton clothes The fingers of the left hand are flexed, but can be passively extended. The left hand is much smaller than the right. Thenar and hypothenar musculature gone. The left wrist can be extended and flexed. The musculature of the left forearm is wasted, and to a lesser extent of the left arm.

The left lower extremity is about three inches smaller than the right. The musculature of the left foot, calf, peroneal group as well as the quadriceps of the left thigh is almost entirely wasted. The flexor muscles of the thigh are fairly well preserved. The gluteal muscles are wasted and the gluteal fold is absent. The small muscles of the left foot are almost entirely wasted and the toes cannot be moved actively but are moved passively. The left thigh is bent forward anteriorly. There is right hypotonia. Curvature of knee backward. The muscles of the right foot also are beginning to atrophy from below upward. The abductors and adductors of the left thigh are intact.

No scoliosis but a lordosis is present with the convexity to the right. The scapulæ are winged. The left supraspinatus is intact. The left triceps and biceps are beginning to atrophy. The flexor and extensor muscles of the left arm are fairly well atrophied as well as the small muscles of the left hand. Beginning atrophy of the small muscles of the right hand and forearm. Those of arm are well preserved.

No sensory disturbances. No reaction of degeneration. Reflexes on the left side are absent; on the right side diminished. No enlarged glands.

Hypertrophic Type.—F. J., 18 years; single; German; in U. S. 15 years.

F. H. Parents healthy, first cousins. Mother had three miscarriages, first three pregnancies, One boy suffers from similar disease; two other boys healthy. One child died of summer complaint. No tuberculous, nervous or syphilitic history. (Fig. 9.)

P. H. Fourth child. Breast fed. Measles at 6 years. Began to attend school at 7 years. Menstruated at 13, regular.

P. I. Began 11 years ago. Teacher first noticed, when she was 8–9 years old, that she walked stiffly with the left leg. At about that time she had difficulty in going upstairs. Gradually grew weaker and weaker until walking on a level became difficult. The slightest push would throw her over. At 15 she was totally unable to walk. The left lower extremity was always weaker than right. Six years after onset of initial symptom, *i.e.*, at about 14, began to complain of weakness in the upper extremities. It was difficult for her to lift them, and gradually these were involved in the same manner as the lower extremities. For the last two years has been unable to sit alone, though she can be propped up in a chair, and is completely helpless; she cannot raise her arms or change her position in bed, but is able to use the fingers.

Rectum and bladder normal. No pain or paresthesias in any of the four extremities. No headache. Sight, hearing and smell normal.

Status.—Female, of average height. Face: Features apathetic, full, fat, typical myopathic. Mouth has lost elasticity and compressibility. Can wink with one eye, wrinkle forehead, pout lips and whistle. Cannot bring the incisors together, and says she never could do so. Molar teeth abnormally large.

Patient in bed: While lying rests on shoulders and buttocks; marked lordosis of spine; back typical crescent shape, the highest bend being at about the twelfth dorsal vertebra. Cannot move from side to side. The entire lower extremities are covered with hair, assuming the male appearance. The feet are in a position of extreme extention and eversion, which, however, can be easily reduced. Legs very voluminous. All the tendon reflexes are absent. Abdomen stout, pendulous. Condition of viscera cannot be ascertained on account of the enormous

thickness of abdominal walls. Skin dark, shows no abnormalities. No enlarged glands. Heart and lungs normal.

Muscles.—The muscles of both shoulders are totally atrophied, and patient is unable to lift shoulders. Where the supra- and infraspinati are normally situated, two distinct, hard movable patches can be felt, which feel like lumps of adipose tissue. Both shoulder joints are loose and there is a subluxation. Patient can move elbows. On deep palpation of the lower part of both arms the tendinous cords can be felt, but these, instead of merging into the various muscles, are continued with paths of adipose tissue. When the arm or forearm is raised, patient cannot alter position, and limbs are entirely under the influence of gravity. The flexors and extensors of the wrists and digits are apparently intact, for the patient can extend wrists and fingers. Cannot raise herself from bed. The muscles of the back are almost all wasted. The wasting of these muscles produces a well-pronounced deformity of spine so that when the patient is in sitting position her trunk telescopes into the pelvis. See Fig. 4. The muscles of the neck are intact. Is able to move head in all directions. The glutei are apparently degenerated. All of the various movements of the lower extremity, flexion and extension of the toes, etc., remain intact, all others are abolished. Both lower extremities are entirely under the influence of gravity. The abdominal muscles are only partly involved. Breathing mainly costal. On deep palpation of abdomen, however, contraction can distinctly be felt.

Facio-scapulo-humeral type.—S. J., brother of the above patient, 13 years old.

P. H. Eighth pregnancy. Began to walk at 15 months. Was never sick when a child.

P. I. Onset dates back six years, *i.e.*, when 7 years old. Teacher noticed that he had difficulty in mounting stairs, running, etc. A few weeks after onset, walking upstairs became more difficult and finally he had to be carried. He became more uncertain in walking and would fall easily. Finally he was unable to walk on a level. Some difficulty in voiding urine. No pain or paresthesiæ. No facial involvement. Upper extremities not affected until $10\frac{1}{2}$ years of age. Unable to turn in bed.

Status.—Patient large for his age. Nutrition fair. Muscles

FIG. 10.—Facio-scapulo-humeral type of primary myopathy.
(Collins)

FIG. 11.—Typical attitude and station of a patient in the early stages of primary myopathy.

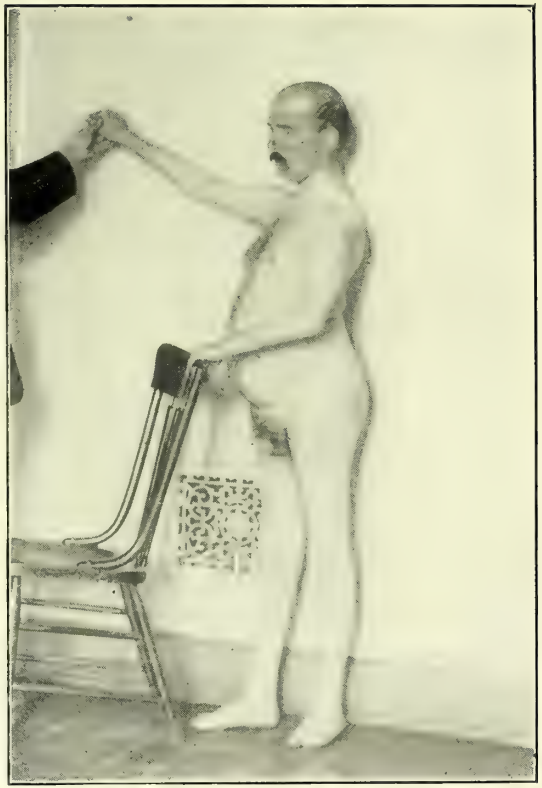

FIG. 12.—Juvenile myopathy difficult to distinguish from chronic poliomyelitis.

flabby and weak, all the muscles below neck being affected. There is profound lateral curvature and scoliosis; the convexity being toward left causing marked deformity. Bulging of left chest internally. Joints all prominent. Talipes equino-varus. Cannot stand due to weakness of the trunk muscles. Body somewhat straightened out by support on shoulders.

Upper extremities: Atrophy of infra- and supra-spinati muscles on right side. Both shoulder girdles enormously atrophied. Atrophy and great weakness of pectorals. Latissimus dorsi well preserved. Subluxation of shoulders. Joint of right humerus enlarged. Bones very thin, middle of humerus almost as thin as a pipe-stem. Bones of forearm and the femur are also atrophied. Ribs very large in contrast. Biceps atrophied. Finger joints normal. Thenar and hypothenar wasted. All tendon reflexes absent.

Lower extremities: Wasting of gluteal muscles and some tendency to contraction. Hamstrings show partial atrophy and loss of power. Loss of power in the abductors of buttocks. Atrophy equal on both sides. Femora atrophied. Knee-jerks apparently normal. Tibiæ normal. Feet: Talipes equino-varus. Tendon reflexes absent. Sensation normal.

Face: Loss of expression. Naso-labial fold very indistinct Slight tremor of lids. Pupils react. Eye-lids can scarcely be closed and when closed the slightest pressure suffices to open them.

Muscles of neck normal. A symmetrical bulging of left, sinking of right. Scapuli flaring and move on respiration. Heart normal. Lungs: Respiration 20, shallow. On percussion, hyperresonance over right anterior. Ptosis of all the viscera, mainly to left. Recti abdominis muscles weak. (Fig. 10.)

Atrophic Type.—E. B., boy 9 years old, U. S.

F. H. Father alcoholic, suffers from asthma, and has had attacks of insanity. Father's sister insane. Mother apparently well. First pregnancy resulted in miscarriage. One child died of unknown cause. Of the six children living three are affected with dystrophy, all boys. This patient is the third child. (Fig. 11.)

P. H. Born easily. Was a healthy baby. Had measles and scarlet fever three years ago. Began school at age of 6 years. Was bright mentally and made fair advancement.

P. I. First symptom was noticed at 7½ years of age. Parents remarked that while walking upstairs he supported his body on thighs with his hands, and also that he stumbled frequently. Six or eight months later he began to walk on tip-toes. From this time he rapidly grew worse and his shoulders began to be thrown back. The family physician attributed this to a habit of the child. Patient gradually became thinner, especially in legs. He never lost the power to use hands.

Status.—Boy slightly underdeveloped for his age. Nutrition good. Adipose moderate in amount. Double equino-varus. Myotatic irritability absent. Station good. Typical "rooster" gait. Marked lordosis of spinal column, with shoulders thrown far back and chest forward. On attempting to arise from recumbent position undergoes a series of movements typical of dystrophy. Some cervical, axillary, and inguinal nodes are palpable. Adenoids present. Teeth: Upper two cervical incisors large in proportion to the other teeth, notched and somewhat corrugated. Movements of head and neck normal. Facies des sphinx. Post-spinal muscles moderately atrophic. Marked wasting of pectoralis and latissimus dorsi, and to a lesser extent of biceps and triceps. Tendon reflexes markedly diminished. Superficial reflexes present. Extremities cold and cyanotic. Testicles undescended. Calves and musculature of thighs atrophied. The thighs, six inches above upper border of patella, are 12 inches in circumference on each side.

Type of Contractures.—J. L., 13 years, 9 months.

F. H. Father, as well as patient's brother, give indefinite history of lues. Mother suffers from pains in legs. Mother's brother insane. One uncle died of tuberculosis. Six children living and well. Fifth pregnancy resulted in miscarriage.

P. H. Patient one of twins. Other twin has weak heart and complains of pain in extremities. Birth normal. Backward in walking. Not as bright as other twin in mind or action. Measles at 9 months, complicated by pneumonia, high fever, no convulsions.

P. I. Healthy up to 5 years. After exposure he was very sick with high fever and coma lasting two days. Attended school until 7 years. At about this time family noticed that he did not walk as he used to and did not run about like other boys. Also noticed that both scapulæ flared. Easily fatigued. Since illness

of five years ago he has gradually become thinner, and his present illness apparently began at that time. He was easily thrown down in the street. After falling at one time was unconscious and had several fainting spells following this, which came on after any excitement. Since then his gait has become worse. Always had enuresis and often had to be excused from schoolroom. Frequent urination, 20–30 times daily. For the past three years the upper extremities have been weak and stiff, so that patient cannot comb his hair. No paralysis. Patient states that he has always seen double. Disposition always quiet, melancholic and nervous. Headache on arising. No rickets. Always had choreiform movements.

Status.—Medium height, fairly well nourished. The shoulders and arm appear small. Stands on toes, heels rest about two inches from floor (right higher than left). Any attempt to put the heels on floor causes him to fall backward. Stands with broad base, feet turned out. Shoulders thrown back and abdomen forward. Gait distinctly equinus. Raises the feet high and then slaps them down on the floor.

Head brachiocephalic. Ears, large, red, flaring. Eyes: slight tremor of lids. Pupils equal and react. Both discs pale. Arteries prominent. Contour normal. Slight nystagmus in external positions. Visual fields normal. Mucous membrane of lips bleed readily; gums spongy. Teeth poor, irregular and degenerate. Tongue protruded straight, shows indentation of teeth; coarse tremor. Palate high, narrow, arched. Uvula long. Tonsils large, congested and slightly edematous. Reflexes present. Flexion of head not possible; lateral movements restricted. Neck rather long. When the cervical spine is steadied the head can be turned from side to side and forward and backward, no flexion being possible, and he is prevented from making the slightest bowing motion. Carotids pulsate visibly on both sides. Atrophy of sterno-mastoid. Sternoclavicular parts evidently separated. Muscles of upper chest atrophic; pronounced atrophy at shoulder girdle. Clavicular part of pectoralis atrophic. Scapulæ flaring, left more than right. Heart: Apex beat visible; also slight epigastric pulsations. Pulse 72. Arteries not thickened. Systolic murmur; rough second sound.

Additional Status.—(Now 14 years old and no signs of puberty).

Under-sized, fairly well nourished. When standing, in order to have one foot completely in contact with floor, the knee of other (right especially) must be bent, and the heel of other foot raised from floor. The right foot is in a state of pes planus. He stands on toes, resting about two inches from floor (right higher than left). In walking he lands on the ball of each foot, and at the same time clutches the floor with flexed toes. The heel of right foot never touches floor, while that of left barely approximates it. Action at knee-joint is that typical of drop-foot. In turning he turns on tip toes. The weakness of extensors responsible for this gait is best shown when he attempts to squat. In proportion as his pelvis goes towards the ground, the heels come forward so that when he is in a complete squatting position he is on tip toes. When at rest, both feet are in a position of complete foot-drop. The knee and ankle jerks are both present. The rigidity of the head and neck and the upper part of the back is so pronounced that the position of the body is like that of spondylosis, but this is compensatory. Evidently a " sport " or sporadic case.

Juvenile Type.—An unmarried paper-box maker, 34 years old. At the age of 31 he began to complain of weakness in the knees, which gradually increased so that he often fell, usually backwards. About 18 months after this symptom came on, his friends remarked his peculiar gait. He swayed from side to side, and about this time he noticed a weakness of the left arm, and others noticed a change in the facial expression.

The most interesting information about this patient was obtained many years after he came under observation, and that was that a sister had a similar disease. The status of the patient and the characteristic features of the disease are best illustrated by the accompanying illustration (Fig. 10).

SUPPURATIVE LESIONS OF THE PHARYNX.

BY CHARLES GRAEF, M.D.
Instructor in Diseases of the Nose and Throat, New York Post-Graduate Medical School and Hospital.

Of the conditions properly included under this head, that most commonly met with is "*peritonsillar abscess*" *or quinsy*.

In no other acute throat trouble is precision in diagnosis and treatment so fully rewarded by relief of distress as in this, and yet these cases are among those most frequently left to worry their way through a painful and tedious course to a convalescence shadowed with the hopeful prognosis that, possibly, a similar experience may not again fall to the lot of the sufferer for some time.

This state of affairs is partly due to a mistaken conception of the morbid changes present in cases of quinsy, and partly also to timidity or uncertainty, on the part of the practitioner, as to the necessary measures of treatment. The name *quinsy* is said to be derived by misuse from the term "*cynanche*", a word commonly applied at one time to all throat conditions associated with pain and obstructive swelling of the parts. This may explain, in a measure, the diversity of ideas still associated with the term "quinsy." Among the answers received from the members of a group of practising physicians to whom the question was recently put were illustrations of these varying ideas:

(a) Membrane formation in the throat is associated with the condition known as quinsy.

(b). Quinsy is a severe inflammation of the tonsils, with incidental swelling of other pharyngeal tissues.

(c) Quinsy is an inflammatory disease of the pharynx and larynx.

The name "peritonsillar abscess" is much the better term to apply to this condition, for it defines very clearly the seat and nature of the lesion—a purulent inflammation of the tissues *around* the tonsil. In most instances the tonsils share only passively in the picture presented, although it is probable that in all cases the organisms responsible for the infection use the tonsil as a pathway of approach to the seat of disease.

Bosworth and others insist strongly that a rheumatic taint in

the individual is chiefly to blame for attacks of quinsy, and if one is willing to accept the modern theory that rheumatism is due to unhealthy tonsil conditions which furnish the avenue, if nothing more, by which rheumatism reaches the body, the relation of quinsy to rheumatism seems settled. That some cases of quinsy can be aborted if taken in hand early—a fact for which the writer will vouch from personal experience—and by remedies ordinarily regarded as *antirheumatic*, is the strongest argument that can be advanced to support the idea. Aside from this it is to be remembered that, in the temperate zone, rheumatism is a very common trouble, and comparatively few patients fail to give a history suggestive of rheumatic affection of some sort. Personal observation convinces me that in a considerable percentage of cases of quinsy the throat condition is the only manifestation that could be called rheumatic, even with this theory in mind. Much more can be done for those subject to attacks of quinsy by bearing in mind the importance of the local causative factors and ignoring, or dwelling but lightly upon, what is but a theoretical explanation.

The practitioner who keeps the "rheumatic" idea too much to the fore in approaching these cases, is most likely to have the unhappy patients already spoken of. Unhealthy tonsil conditions can confidently be considered the chief cause of all cases of quinsy, and in most of them the connection is easily demonstrated.

Abscess formation in quinsy cases may occur at any point surrounding the tonsil. The seat of election is the anterior part and in the soft palate, arching upward from this; these pus collections generally "point" somewhere between the base of the uvula and the center of the anterior faucial pillar. The cellular tissues below and behind the tonsil, however, are frequently the places where the pus collects, the posterior pillar of the fauces instead of the anterior showing the bulging. The chief seat of invasion may be above the tonsil and to its outer side, in which event the angular recess known as the "supratonsillar fossa" often gives egress to the pus.

In all these cases the tonsil is pushed from its proper relation to other parts of the throat. It moves inward, forward, upward or downward, as the case may be, in obedience to the pressure from the swollen areolar tissue in which it rests; an edema of

the palate and especially of the uvula commonly occurs, and hasty inspection may easily lead to a diagnosis of inflammation in the tonsil itself; this is all the more likely because of the difficulty the sufferer has in opening the mouth to permit thorough examination. For these reasons the stab puncture, often relied upon as a relief measure, is frequently of no avail. This also explains why some attacks are so much more distressing than others. Most cases of quinsy are confined to one side, but bilateral cases are not rare. They are usually more severe in this event.

As an abortive agent, at an early stage, no other drug has been so effective in the writer's hands as *guaiacum*. The ammoniated tincture may be given in from one to two dram doses, according to the age of the patient, in a glass of milk, or, if preferred, in an emulsion with egg-albumen or mucilage. The dose should be repeated every two hours until the pain disappears, unless gastro-intestinal irritation contraindicates its continued exhibition. Salicylate of soda is favored for the same purpose, and with this may be combined the local application of the bicarbonate, the powder being smeared at frequent intervals over the affected parts by the patient, with moistened finger-tip. General measures for the comfort of the patient, good nursing, liquid diet, etc., are necessary.

If, as commonly happens, the case is seen too late for such treatment, careful examination should be made to locate the pus collection, and when this is determined it is the duty of the medical attendant to make a free opening for drainage. An ordinary straight or curved bistoury may be used for the purpose. A shank to prevent the knife penetrating too deeply is easily provided by wrapping a few layers of adhesive plaster around the blade, about three-fourths of an inch from the point.

In the type of case usually met with, which is illustrated in the accompanying drawing, the incision should begin about half an inch from the margin of the anterior faucial pillar, and a little above a point in line with the lower molar teeth. The cut should extend upward and inward in a curved direction, parallel with the edge of the pillar, ending at a point half an inch behind the base of the uvula. The direction and extent of this incision may be seen in the drawing. If this incision is properly made, it will always release the pus. Contrary to general belief, hemorrhage is comparatively slight.

The pus in cases of the other types mentioned must be sought for at the most probable point in each instance, and it is in them that the following plan of approach is especially helpful, for not infrequently search for the pus with a bistoury is difficult, and may become dangerous. For this purpose a pair of long, slender, but strong, angular forceps is necessary, the narrow point of which is to be pushed into the tissue at the selected place.

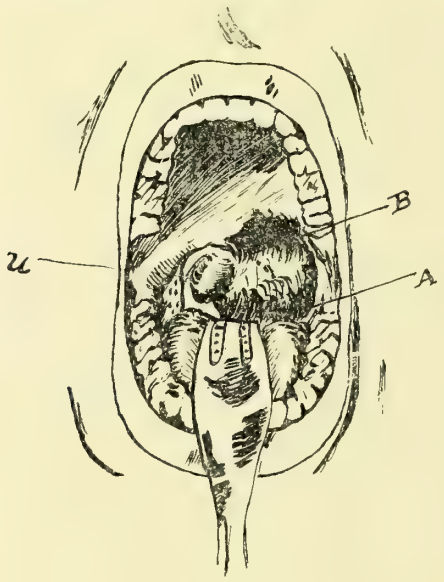

Fig. I.

A rough sketch of a typical quinsy on the left side. The swollen uvula, *u*, is pushed over by the abscess in front and above the left tonsil. The line A to B indicates the extent and direction of the incision which will best serve the need for drainage in these cases.

The blades of the forceps are then opened, by which a considerable tear can be made, through which the pus will drain. This method of opening the abscess may be used instead of the bistoury, even in the first type of the disease if circumstances make it desirable, but a better and cleaner incision results from the use of the knife in this region. The tear produced by the forceps, besides being easy to make, has less tendency to bleed than a knife cut.

The free openings thus made have little or no tendency to close before the abscess cavity is clean, and when successfully made insure rapid subsidence of the disease. When thorough drainage has been established, cure may be hastened by a gargle of peroxide of hydrogen diluted with boiled water—one part to four. This gargling should be repeated at intervals of an hour or even half-hour. An iron mixture may be prescribed with advantage in most cases. Of these, none is better than a suitable dose of the tincture, dispensed with glycerine, and directed to be taken in half a glass of effervescent soda water.

In all cases recurrence is best guarded against by careful, intelligent attention to the throat. There need be no hypertrophic tonsils; often there is little of any sort of tonsil showing, but search in this region nearly always reveals unhealthy tissue. Quite often this will be found as an infected mass buried within

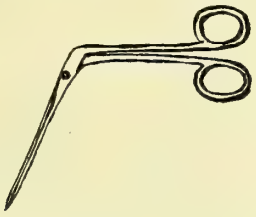

Fig. 2. Angular forceps for opening peritonsillar abscess.

the mucous membrane—the so-called *submerged tonsil*—or infected lymphoid tissue may lie behind an apparently clean tonsil Search of the recess above the tonsil, called the supra-tonsillar fossa, will often lead to the detection of such a condition. After a throat has withstood one or more attacks of quinsy, the tissues about the tonsils apparently remain more or less saturated with organisms, and even the most skilful removal of the tonsil may fail fully to eradicate the tendency to recurrent suppuration. It is somewhat analagous to the condition of the skin of the neck in certain individuals who suffer from repeated outbreaks of "boils." Change of climate, diet, bathing, and a course of alteratives are necessary for some of these patients.

In infants and young children, abscess formation in the pharynx is not rare, but in them it assumes a special form, and is not in close relation to the tonsil. In these subjects the *retro-*

pharyngeal abcess is the characteristic type. This may be due to the presence in early life of a comparatively deeper cushion of connective tissue under the mucosa of the rest of the pharynx, than that surrounding the tonsils; or, perhaps the abundance of lymphoid elements in the mucous membrane during the period of active growth is responsible. The fact remains that in young persons abscesses in the pharynx are found, as a rule, in the lateral walls of this space, the posterior wall being involved less frequently. With the rare cases due to disease of the vertebrae, this article is not concerned; in the majority of cases, if any special cause is assignable, the abscess is a sequel of one of the diseases of childhood. More often no particular reason for the trouble can be found. Development of such an abscess is commonly insidious. Loss of appetite, restlessness, perhaps cough, are the chief indications that something is wrong. Later, the child develops difficulty in swallowing and may refuse nourishment on this account. Dyspnea, snoring, and breathing through the open mouth are common and may lead to a diagnosis of adenoids.

As the abscess becomes painful and large, the child may hold the head stiff, inclined forward and to the side. The swelling usually occurs in the upper part of the pharynx, and is generally within view. In a small infant this view may be very limited, however, and diagnosis must be made by palpation. The soft, elastic bulging due to such a tumor is easily recognized, and at times the abscess wall is ruptured by the finger-nail while making the examination. If the membrane is too tough for this, a pair of narrow-bladed scissors serves best to open such a tumor. It should be cut at a dependent part and may then be opened wider by separating the blades of the instrument in the wound. It is well immediately to hold the child with the head downward, as these pus-sacs are often quite extensive, and dangerous symptoms may follow discharge of the contents into the larynx.

The after-treatment should be based on the general condition of the child—fresh air, nourishing food, tonics, cod-liver oil, etc. A second opening of such an abscess is seldom necessary, but may be made if the tumor recurs. Similar abscesses may occur in adults, but such instances are rare.

I recently had occasion to open a large abscess at the base

of the tongue, in the region of the lingual tonsil. It occurred in a young man of 19 years, who suffered with hypertrophic turbinals, but was otherwise healthy. This abscess may have been due to injury, as by a fragment of bone, though no such history was given.

The condition known as " acute phlegmon of the pharynx," which occurs in adults, and of which occasional cases are reported, is an exceedingly virulent form of infection in which a fatal outcome is to be expected. The writer has had no personal experience with this condition; but a recent case of phlegmon, due to injury of the pharynx by a bone, illustrates very well the danger of attempting treatment of throat conditions without an adequate view of the parts involved.

The patient was an old man, weighing about 350 pounds. He had the ill-luck to get a fragment of bone stuck in his pharynx. He sought the help of the nearest physician, who attempted to clear the obstruction by pushing the fragment down to the esophagus with a " brush " or " bougie " made for this purpose. Instead of succeeding, he evidently pushed the bone through the wall of the pharynx. I saw him some days later, and found a swollen cushion of mucous membrane extending from the lower part of the left posterior faucial pillar to the side of the larynx. I incised this swelling at several points, but was unable to find the bone spicule. It seemed possible that the throat had suffered only a scratch from the passing bone, which had, perhaps, been safely pushed down by the brush. This was apparently borne out by the marked improvement following local treatment with adrenalin and peroxide of hydrogen. A week later an abscess was found in the pharynx wall, which became quite extensive. As I did not believe this patient would survive the operative treatment necessary to explore the diseased parts, I declined to undertake it. He was, however, operated upon by a very skilful surgeon. The spicule of bone was discovered and the abscess drained, but the patient died the next day.

The plan employed by the physician who first saw this case has long been taught in standard works on surgery, but it is archaic and the experience proves the necessity of avoiding any manipulations about these parts that cannot be correctly guided throughout by observation with a laryngoscope.

GAS SEPSIS, EMPHYSEMA, OR PHLEGMONE; TWO RARE CASES.

BY WILLIAM HENRY PORTER, M.D.

Professor of Medicine, New York Post-Graduate Medical School and Hospital.

These two cases of general gaseous or phlegmonous emphysema are reported on account of their rare occurrence.

The first case came under my observation at the necropsy table in the year of 1880.

The history was that of a woman about 35 years of age, a chronic alcoholic subject, who suffered also from a diffuse form of nephritis. The patient died rather suddenly and without apparent cause. At the necropsy, which I performed a few hours after death, the following most unusual conditions were noted. The body as it lay on the necropsy table was bloated to the utmost capacity of the skin. The distention, however, was not edematous in character, but crackled under pressure as if there was a large accumulation of gas bubbles in the subcutaneous tissue. The skin covering the head, neck and face was so much distended that the head appeared like a great round ball, attached by a very short thick stem to the very much distended trunk. The anterior surface or facial side of this ball-like extremity was marked by a decided opening, as it were, in the skin, which represented what had been during life the oral orifice; all the other facial markings were obliterated. When the integument was divided, bubbles of gas escaped from the cut surface. All the internal organs and the areolar planes were filled with gas bubbles, giving every structure examined a distended and emphysematous appearance. All the organs of the body crepitated when pressed upon and gave off gas bubbles from their cut surfaces. All the fluids of the body were frothy. There was no surgical or marked antemortem condition noticed by the physician in attendance that could have accounted for this extensive formation of gas. A slightly emphysematous condition was observed by the attending physician in the region of the neck just before death. This condition progressed, however, with great rapidity during the few hours that elapsed between death and the necropsy. Aside

from the general emphysema the only anatomical lesion found at the necropsy that could in any measure explain the peculiar emphysematous condition was marked gangrene of both kidneys.

In the light of our present knowledge the gangrene and emphysema are the results of a general infection. As this case was observed in 1880, long before the development of our modern conceptions regarding the gas-forming bacilli, no cause was discovered that could adequately explain the extensive gangrene in that locality or the associated diffuse emphysema.

The second case observed occurred in a farmer, aged 40, married, who was admitted to my service, in the Post-Graduate Hospital, May 12, 1904. Up to six months prior to entering the hospital he had always been strong and well. During the six months prior to admission, he complained steadily of illness; in fact, he grew worse continually during this period. The chief symptom complained of was pain in the region of the bladder and right kidney. At first the pain was mild in character, but gradually grew more intense and was not materially modified by treatment. It was most severe in the region of the bladder, radiating upward along the line of the ureters. Prior to admission to the hospital he had frequently suffered from intermittent hematuria. He was catheterized prior to admission, but so far as could be ascertained there was no retention, and no definite diagnosis had been made.

In spite of treatment the pain grew worse steadily, and was attended with progressive loss of appetite, flesh and strength. When admitted he was so weak that it did not seem wise or humane to subject him to a thorough examination of the genito-urinary tract. On auscultation the muscle tone of the heart was found to be extremely weak, and the sounds of the heart very indistinct. The physical signs relative to the pleura and lungs were normal, with the exception of a few superficial friction sounds, heard on the right side, both anteriorly and posteriorly. These friction sounds were interpreted as indicative of old pleuritic adhesions. Palpation over the region of the right kidney showed slight enlargement of that organ. A decided tumefaction, apparently connected with or located within the cavity of the bladder, was made out in the hypogastric region. Percussion over this mass elicited pronounced

dulness, which extended from the region of the bladder upward along the line of both ureters as far as the kidneys, most marked on the right side. Palpation greatly aggravated the pain, and for this reason the interior of the bladder was not explored.

On May 13 the patient voided urine without the aid of a catheter. It was violet in color, spec. grav. 1.010, acid in reaction, and quite fluid when first passed, but on standing it became thick and viscid, apparently with a mucus-like substance. It contained a trace of albumin, but no glucose, and 2% of urea. No indican. No blood or casts, but a few leucocytes and squamous cells were found on microscopic examination. There were no crystals, and no fragments of tissue indicative of a new growth in the bladder could be found. In fact, the urine furnished little or no information. On the other hand, the objective symptoms suggested a process that was rapidly exhausting the patient's vitality. The pain and the tumefaction in the hypogastrium pointed strongly to a malignant growth in the bladder.

The pulse, temperature and respirations were in general above normal, as recorded in the accompanying chart (see Fig. 1.)

Shortly after admission to the hospital the patient was given several small doses of calomel, which acted freely, after which he suffered less pain and remained fairly comfortable during the two succeeding days. The temperature and pulse receded a little, but the patient lost in strength; therefore no thorough examination of the genito-urinary tract was attempted during his stay in the hospital. Two days later, after having passed a very comfortable night, the patient became somewhat drowsy, alternating with a mild delirium. During that day he complained in his lucid intervals of severe cramps in the calves of the legs, and he had several involuntary stools, with one of which considerable blood was passed. Whether the blood came from the bowel or with the urine, which might have been voided at the same time, was not determined prior to death. In the light of the necropsy findings, however, it is highly probable that the blood came from the bladder. At all events he failed rapidly after this hemorrhage, the pulse became more feeble and at times almost imperceptible. He gradually passed into a comatose state and died on the afternoon of the fourth day after admission. Just before death a slight emphysematous condition was noted on the right side of the trunk and neck anteriorly.

At the necropsy, held about 16 hours after death, the following conditions were found:

There was a marked area of subcutaneous emphysema on the anterior and right side of the chest near the root of the neck. Rigor mortis was general and well marked. The body was moderately emaciated. When the integument and subcutaneous tissue were divided, gas bubbles escaped from the cut surface. Upon opening the abdominal cavity there was an escape of a large amount of foul-smelling gas.

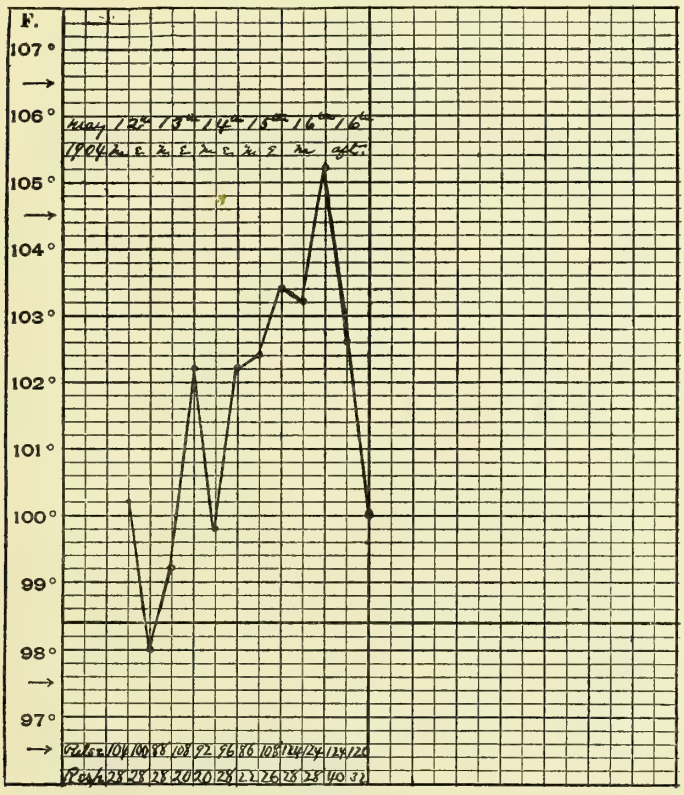

FIG. 1.

The *heart* was soft and flabby and seemed to have stopped in diastole. There was a slight, so-called antemortem blood clot both in the aorta and ventricular cavities. The valves were normal.

The *left lung* was free from pleuritic adhesions. On incision an unusually large amount of gas escaped, and some of the gas bubbles apparently escaped from the cut surfaces of the vesicular walls as well as from the air sacs The left lung contained a number of calcareous

tubercle-like nodules, evidently marking the former seat of imperfectly resolved areas of bronchopneumonia. Upon the right side were numerous p'euritic adhesions, especially marked anteriorly near the right border of the sternum, but also quite numerous about the apices both anteriorly and posteriorly. The lesser fissure was practically obliterated by pleuritic adhesions. The *right lung*, like the left, was unusually emphysematous, giving off numerous gas bubbles from the cut surface. The blood exuding from the divided ends of the blood vessels was filled with gas bubbles. There was little or no evidence of edema of the pulmonary tissue in either lung; in fact, they were, so to speak, nearly dry.

The *spleen* was slightly enlarged,soft, very dark in color, and very emphysematous, and upon pressure crepitated almost like lung tissue. Gas bubbles escaped from the cut surface, and a very frothy, blood-tinged fluid escaped on compression.

The *bladder* was distended and contained a frothy fluid. The wall was about two centimeters (0.78 of an inch) in thickness, due to a diffuse carcinomatous infiltration of the muscular coat. Within the bladder was a diffuse papillary growth, which is well shown in the accompanying cut (see Fig. 2). Microscopic examination of the growth proved its carcinomatous character: papillomatous within the cavity of the bladder, schirrous in the wall and underlying structures. The lumen and wall of the *ure'ers* contained innumerable gas bubbles, producing an appearance similar to what is seen in cases of ascending hydronephrosis (Fig. 2). There was no fluid in the lumina of the ureters, however. The tissue of the right ureter also was considerably infiltrated with a schirrous-like material, which made the right tube much harder than the opposite one. The left ureter was quite soft and much more distended than the right tube. A similar difference existed in the substance of the two *kidneys*. While both were enlarged and contained an abundance of gas bubbles, the left was the larger of the two; the right kidney, on the other hand, was firm, somewhat hard, as if infiltrated with a dense substance and also distended with gas. The left kidney (see Fig. 2) was by far the larger of the two. Both organs were in a state of parenchymatous metamorphosis.

The *liver* was free from adhesions and about normal in size. There was one small metastatic growth in the liver, almost white in color, contrasting strongly with that of the hepatic tissue, which was unusually dark, almost black. Upon microscopic examination this neoplasm was found to be carcinomatous. The cut surface gave off large quantities of gas bubbles. The *gall-bladder* was well filled with gall-stones and bile, and slightly adherent to the under surface of the liver and to the hepatic portion of the colon.

The *colon* was apparently empty and practically collapsed. The appendix was normal. The *small intestine* and *stomach* were both unusually dark in color, considerably distended with gas and contained throughout a frothy fluid. Free gas bubbles appeared to be present in the walls of the intestine. Although there were a few adhesions between the coils of the small intestine, the foramen of Winslow was patent, and

FIG. 2.—Showing the interior of the bladder with its rough, papillary-like surface; dilated ureters and enlarged kidneys. *a*, thickened wall of the bladder, infiltrated with carcinomatous tissue. (Porter)

there was no evidence of intestinal obstruction at any point. The *pancreas* also was decidedly emphysematous; otherwise, nothing abnormal was found. The *blood-vessels* of the abdominal cavity were distended with blood and gas, largely the latter. The *spleen*, *liver*, and *small intestine* looked as if they were about to break down into a gangrenous mass, yet no decidedly gangrenous focus could be found. The *brain* and *spinal cord* were not examined.

In this interesting case of carcinoma of the bladder, complicated by a very pronounced emphysema of the ureters, kidneys and all other internal organs, and the areolar planes of the body, we have one of those rare instances in which a large amount of free gas is found at the necropsy in the various organs and tissues, all of which had occurred in the absence of any gangrenous or putrefactive condition that could have accounted for the emphysema. In the first case there was a still more abundant accumulation of free gas in all the organs and tissues of the body. In this instance the emphysema evidently began to develop without apparent cause shortly before death, and progressed very rapidly, so that at the necropsy, which was made only a few hours after death, all the organs and tissues were extremely infiltrated with free gas. Associated with the extensive emphysema was a very marked gangrene of both kidneys. The latter condition could not have existed for any length of time prior to death, and, in all probability, went hand in hand with the emphysema, the two conditions having a common origin.

There are three varieties of microörganisms that are well recognized as capable of causing considerable gas formation when they become active in the animal tissues and organs, or are grown upon suitable media. These are the *Bacillus edematis maligni*, first discovered and described by Pasteur[1] in 1875 under the name *Vibrion septique*, but more fully investigated and described under its present name by Koch;[2] the *Bacillus coli communis*, isolated from the human feces and described first by Emmerich[3] in 1885; and the *Bacillus aerogenes capsulatus*, discovered by Welch[4] and fully described in an article by Welch

1. *Bull. Acad. Med.*, 1877 and 1844.
2. *Mittheilungen aus dem kaiserl, Gesundheitsamte*, I, 53.
3. *Deutsche med. Wochenschrift*, 1885, No. 2.
4. *Bull. of the Johns Hopkins Hospital*, July and August, 1892, vol. viii., No. 24.

and Nuttall, in 1892. The two former are less prolific in the production of gas bubbles than is the latter. While the *Bacillus edematis maligni* is known to be capable of producing more gas than the *Bacillus coli communis*, there were no macroscopic changes found in either case that would warrant the diagnosis of a malignant form of edema. In fact, both cases were free from edema. Further than this, the *Bacillus coli communis* never produces such large quantities of free gas all through the body in so short a period of time; therefore, it can be excluded as an etiological factor in these cases. By this process of exclusion it seems reasonable to regard the extensive emphysema found in both instances, and the gangrene in the first, case as due to inoculation just before death, with the *Bacillus aerogenes capsulatus*.

In the first case observed, the diagnosis could not be determined by a bacteriological standard, because it was not until 1892, when Welch recorded his discovery and gave us a full description of the bacillus, that the pathology of this particular class of cases began to be correctly understood.

Unfortunately, no bacteriologic examination was made in the case of carcinoma of the bladder with general emphysema. Nevertheless, it is highly probable that both these cases were due to a gas-producing bacillus, probably the *Bacillus aerogenes capsulatus*; for this microörganism is known to be widely distributed in nature and can easily gain access to the system. It is also known to be a very prolific producer of large quantities of gas in a very short space of time after death, and in the absence of all other conditions, that might be made to explain the diffuse emphysema.

Prior to the discovery of this particular bacillus of Welch, cases of this nature, and especially those occurring in the absense of any ante- or post-mortem decomposition, were attributed to the entrance of air into the circulation, even though no well-defined cause or portal of entry could be discovered. In neither of these cases was there any ante- or post-mortem condition discoverable that could logically be made to account for this large accumulation of gas throughout all the tissues and organs of the body. Even in the first case, as already stated, it is highly probable that the gangrene of the kidneys was caused by the action of the bacilli, and that it did not act as the cause of the emphysema.

Welch[1] in his Shattuck Lecture before the Massachusetts State Medical Society, in 1900, argues that "it is not possible to explain some of the cases in which bacilli and gas have been found in the heart, blood-vessels and organs very soon after death, especially when the corpse has been kept in a cool place, otherwise than upon the assumption of the distribution of the bacteria by the circulating blood." In like manner in these cases, assuming that they were produced by this form of bacterial infection, it is difficult to account for the conditions found except through infection of this character and a distribution of the bacilli by the circulating blood just before death. The commencement of the emphysema in the areolar planes of the neck, which was noticed in both cases just before death, sustains this contention. In the latter instance the presence of the gas bacillus of Welch was not demonstrated to be present, as should have been done to establish the diagnosis beyond a question of doubt; nevertheless, these two cases are so pronounced that they seem to be worthy of record, and probably both had this bacillus for their cause; hence the diagnosis.

The fact that a pronounced neoplasm was located in the bladder, which probably had an eroded surface before death, and the emphysema so pronounced in connection with the ureters and kidneys, renders it highly probable that infection took place along the line of the genito-urinary tract, possibly as the result of catheterization. It is possible also that the portal of entry in this second case was through the intestinal wall and not through the genito-urinary tract. In this connection it is well to note that Welch[1] states that the urinary tract is one of the portals of entry for the gas bacillus, but that up to 1900, the bacillus had not been demonstrated at this point during life, although frequently found here after death.

In the first case, the rapid disintegration of the renal structures indicates that, even if the portal of entry was not by the genito-urinary tract, the greatest amount of damage was found in the kidneys, as evidenced by the extensive gangrene of these organs. The very dark color of the stomach, small intestine, spleen and liver in the second case, indicates that these organs were about to become gangrenous, and that the greatest damage had occurred in these structures. While both

1. *Med. Com. of the Mass. Med. Soc.*, vol. xviii, No. II. 1900.

the genito-urinary and the alimentary tracts are known portals of entry for the bacillus, it is a very difficult matter to determine positively through which channels the system becomes infected. The repeated hemorrhages from the genito-urinary tract in the second case would have a tendency to open the blood vessels and render entrance of the bacilli to the system easy by this route; while in the first case a similar condition might have existed, in the uterine body so far as the blood vessels were concerned, and thus have established the portal of entry at that point.

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AMAUROTIC FAMILY IDIOCY.

Report of a Case with Remarks.

BY A. E. DAVIS, M.D.

Professor of Diseases of the Eye, New York Post-Graduate Medical School and Hospital.

Histologic Report of the Eye.

BY EDWARD L. OATMAN, M.D.

Surgeon Manhattan Eye and Ear Hospital.

This peculiar disease, amaurotic family idiocy, is of special interest to oculists, because of the striking and characteristic changes which take place within the eyes during the progress of the affection, and which are pathognomatic of it.

In fact, it was the observations of these changes in the eyes —consisting of a grayish-white haze surrounding the maculæ, which were cherry-red in color—that led to the discovery of the disease by Warren Tay, in 1881, who published the report of his case in the first volume of the "Transactions of the Ophthalmological Society of the United Kingdom," under the title of "Symmetrical Changes in the Region of the Yellow Spot in Each Eye of an Infant." Tay was of the opinion that the condition was a local affection limited to the eye. To Sachs, of New York, belongs the credit of having first called attention to the general character of the disease. He published a report of a case in the *Journal of Nervous and Mental Diseases* in 1887. H. Knapp examined the fundus of the eyes in this case, and his report is cited by Sachs. To Sachs also we are indebted for the name "*amaurotic family idiocy*," based on clinical symptoms and not on anatomical grounds, as had been done previously, each observer describing the disease under some special name until this time, 1896.

As the disease is a comparatively rare one, and not frequently seen except by specialists—something over a hundred cases having been reported up to date—I venture to cite in brief the chief symptoms of the affection, as given by Sachs himself in 1896:

1. Mental impairment, observed during the first few months of life, leading to absolute idiocy.

2. A paresis, or paralysis of the greater part of the body; this paralysis may be either flaccid or spastic.

3. The reflexes may be deficient or increased.

4. A diminution of the vision, terminating in absolute blindness (changes in the macula lutea, and later an optic nerve atrophy).

5. Marasmus, and a fatal termination, as a rule, about the second year.

6. The occurrence of the affection in several members of the same family.

7. Healthy at birth, remaining so up to the third or fifth month.

Other occasional symptoms in the disease are (1) nystagmus; (2) strabismus; (3) hyperacuity of hearing; in addition to these, Falkenheim has observed two others: "explosive laughter" and "disturbances of deglutition." Disturbances of deglutition was a marked symptom in the patient here reported by me. There was inordinate laughter in the later course of the disease, although in the early stage the child never laughed.

Besides the marked peculiarity in the eyes in this disease, another feature of special significance is its occurrence in several members of the same family, although not necessarily in rotation. The parents are usually healthy, and syphilis has been definitely excluded as a cause of the disease. Another peculiar feature is that, up to the present, the majority of the cases have occurred in Hebrews, usually from the eastern part of Europe. In fact, all the first cases were in Hebrews. This fact was commented upon by Carter; but since his paper, some cases have been reported in other races besides the Hebrew.

The report of my own case is as follows:

Rubin G., admitted to the Babies' Wards of the New York Post-Graduate Hospital, under Dr. Chapin's care, December 21, 1907. He remained until January 21, 1908, at which time he was discharged, but returned in a few days, and died in the hospital February 4, 1908. Father and mother were Russian Jews, healthy. There were five children in the family, three of whom are living and well; two died of amaurotic family idiocy, this case being one of these. The oldest child, which was the other case, was born normally after a labor of only two hours, and was apparently healthy until about the

eighth or ninth month of age, when the mother took the child to Bellevue Hospital on account of a persistent diarrhea. The doctors at the hospital told her that the child was blind. The child was very weak at that time, but the mother thought this was due to the diarrhea. The child died at Bellevue Hospital at the age of 18 months. The fourth child, the patient under discussion, was delivered instrumentally, but in good condition, and was healthy up to about the eighth month, when the mother noticed that the child could no longer sit alone; would not take anything in the hands, or, if it did, would not hold it; never laughed, and apparently was losing its eyesight. It also suffered with diarrhea, as the first child had done. The child was breast-fed, but at the age of eight months refused to nurse. The limbs were flaccid and not spastic; the child never had any convulsions. No history of any such trouble could be obtained from the mother as having occurred in her own family or in that of her husband. Examination of the other children in the family showed their eyes to be entirely normal and the children themselves healthy. (No tuberculosis, no syphilis, no alcoholism, insanity or nervous troubles could be elicited in either the mother's or father's family.)

The condition of the patient on admission to the Post-Graduate Hospital was as follows:

The general paralysis of the limbs had progressed until the child was perfectly limp, except for the fingers and toes, which were slightly spastic. The child was listless and paid very little attention to anything, although the mother gave the history of the child laughing inordinately in the last few weeks. The child's chest was examined, with negative result. The general appearance was only fairly good. The skin was pale, the expression of the eyes vacant, and the baby started at the slightest noise. The child could be fed with the spoon. The bowels were much constipated, sometimes moving only once in a week, and stools resembled "pieces of blood."

This child remained in the hospital from December 21, 1907, to February 4, 1908, with an intermission of a few days, when the mother took the child home. The temperature ranged, during the stay in the hospital, from 98° to 106.3°; pulse ranged from 100 to 144, until the day of death, when it was 164; respiration ranged from 32 to 50, until the day of death, when it

arose to 80. It was difficult to account for the rise of temperature. When the child first came into the hospital it slept fairly well, but later became very restless, often cried out in its sleep, and feeding had to be forced. Difficulty in swallowing in the latter stages of the disease was very marked.

On January 3, I examined the eyes for the first time. The pupils, before atropin had been instilled, were about one third dilated, very sluggish in reacting. Examination of the fundus with dilatation of the pupils showed the typical changes in and around the maculæ, also atrophy of the optic nerve. There was a large grayish-white patch surrounding each macula, while the maculæ themselves were cherry-red in color, horizontally oval. The blood vessels of the retina could be seen very distinctly in this nebulous area surrounding the maculæ, and the optic nerve was intensely white. The blood vessels were about the normal size. The patient was entirely blind. I examined the eyes three or four times later, and at the very last stages of the disease the maculæ became a somewhat brownish red, rather than the intense red as first observed. This change has been noted also by Mary Buchanan in the case she examined for Dr. McKee.

During the stay in the hospital, the child vomited once. Towards the end there was a slight looseness of the bowels. The child became very much emaciated, and died on February 4, 1908. One eye was removed nine hours after death and given to Dr. Edward L. Oatman for histologic examination.

While this peculiar malady has come to be recognized as a distinct clinical type of family disease, largely through the efforts of Dr. Sachs, its etiology still remains a matter of much discussion. Whether the disease is congenital, in the sense that it is due to some inherent defect in the gray matter of the central nervous system; or if the disease is acquired and due to degeneration of some kind, is still undecided.

In all the cases upon which post-mortem examinations have been made there is, on the whole, close agreement as to the pathological findings, and, as Sachs remarks, (1903): " there can be a difference of opinion only in respect to the interpretation of these findings." In Sachs' earliest autopsies, the first in 1887 and the second in 1892, he found evidences of degeneration in the pyramidal cells of the entire cortex of the brain. In the

first case, only the cortex was examined; but in the second, the ganglia, optic chiasm, the pons, the medulla, and the cervical cord were examined. No changes were found, however, in the gray matter of the cord. Later, Kingdon and Russell, in three autopsies, confirmed the findings of Sachs as to the changes in the ganglion cells of the cortex of the brain, and they, like him, were unable to discover any change in the ganglion cells of the cord, the cerebellum, or basal ganglia. The nature of these changes in the cells of the cortex was attributed by Sachs to arrest of development, but Kingdon and Russell thought they were purely degenerative symptoms. Hirsch, in 1898, after a very careful and complete histologic examination, established the fact that not only the cells of the cortex of the brain were affected, but that the ganglion cells of the entire nervous system were diseased. He says that: " we have an equal affection of all the nerve cells of the entire nervous system, the main features of which are a condition of chromatolysis and other degenerative processes of protoplasm, combined with considerable swelling of the cell-body and displacement of the nucleus toward the periphery of the cell. The neuroglia has been found to be perfectly normal, and no affection of the blood vessels could be noticed throughout the entire system."

Sachs (1903), in commenting on the three cases in which he held autopsies—the study of which was much more satisfactory by reason of the improved staining methods than was possible in his first two autopsies—says: " a careful study of these actions show some degeneration in the development of the cerebral white fibres and also a degeneration of the pyramidal tracts in the lateral as well as in the anterior columns of the cord. This same degeneration can be traced through the course of the pyramidal tracts in the inner capsule, crusta, pons and medulla, but much more striking than this, which occurs in the white fibres, is the change in the gray matter of the central nervous system. The changes are found to be the same in the cortex of the brain, in the cranial nerve nuclei, in the anterior and posterior gray matter from the periphery to the lowest lumbar and sacral segments of the cord. Even the sacral ganglia show similar changes. The changes are practically those to which attention has been especially directed by Dr. Hirsch, and which were found to be similar to those reported by me in the cortex, in

1887, and were also found on a re-examination to be continued in some of the spinal sections of the specimen examined in 1892 in my second case." Sachs is also of the opinion that the morbid process in this disease primarily affects the gray matter of the brain and cord and secondarily the white fibres of the anterior and lateral tracts.

The pathological changes within the eyes in these cases are of as much interest to the ophthalmologist as were the clinical findings, which led to the discovery of the disease. Treacher Collins was the first to examine the eyes of such cases microscopically, in two of Kingdon's cases. He considered that the grayish white appearance surrounding the maculæ was due to edema. However, because of the detachment of the retina from the fixation fluid of Müller, the examination proved unsatisfactory and the nature of the change was left in doubt. The third and fourth cases in which the eyes were examined microscopically were respectively those of Peterson and Hirsch (1898), both being examined by Ward A. Holden. In Peterson's case the post-mortem changes were so advanced that the examination was unsatisfactory. In Hirsch's case, however, the examination of one of the eyes was especially satisfactory, and Holden established the fact, for the first time, that the changes in the retina were identical with those in the cortex of the brain and in the cord, and were due to changes in the ganglion cells of the retina.

He says: "Near the fovea centralis where the ganglion cell layer is thickest, the gray color is most intense, and in the fovea centralis, where ganglion cells are wanting, the red color of the choroid shows through, appearing darker by reason of the contrast with the surrounding light gray. . . . The optic nerve atrophy, which follows the appearance of the macular changes, is to be interpreted as presenting both an ascending and a descending degeneration. We have thus found a common explanation of the symptoms of this complex affection and have shown it to be pathologically a unit. Ocular neurons have undergone a primary alteration like the cerebral neurons. Nor should this cause surprise, when we remember that the retina and the optic nerve are genetically part of the brain." Later, Holden's views were confirmed by Shumway and Buchanan (1905), who examined the eyes in the case reported by McKee. Mohr,

however, was of the opinion that the nebulous condition surrounding the maculæ was of an edematous nature, but Falkenheim says the clinical features are against this theory in two particulars: first, the condition of the maculæ remains unchanged for months, and second, there is no veiling of the fine blood vessels in this nebulous area, which would be the case were it edematous in nature.

Etiology.—The nature of the exciting causes, resulting primarily in the degenerative changes in the nerve cells of the entire nervous system, and secondarily in changes in the white fibres, still remains unsettled. Sachs, who has done so much to clear up this mysterious disease, in his first investigation, was of the opinion that the disease was congenital in origin and that the changes were due purely to arrest of development; in other words, was purely an agenetic condition. He reported his first cases under the title of "Arrested Cerebral Development" and "Ageneris Corticalis." Kingdon and Russell were of the opinion that it was an acquired disease and purely a degenerative process, while Hirsch thought it was an acquired disease with degenerative changes due to certain toxins. Sachs claims that the difference between his views and the views of the authors just mentioned is not so great as would appear at first sight. He says: "A degenerative process does not necessarily represent an acquired or acute affection. It is my contention now, as in former years, that degeneration will set in wherever normal growth is arrested, but life continues. A child to be afflicted with amaurotic family idiocy is born with a limited and restricted capacity for normal development. Its gray cells may do as well as any other child up to the age of two, three or six months, but beyond that, its powers for further development will not go. To this extent in this respect, the disease is, after all, a congenital affair; when normal development ceases, degeneration sets in. Gowers has recently written a helpful article in which he applies a similar course of reasoning to the various sclerosest and has suggested the term 'abiotrophy,' which I gladly accep, to indicate this arrest of normal development, followed by degeneration."

The term abiotrophy means simply a lack of vital nutrition. Sachs says if we should accept Gowers' phraseology, we might, with good reason, say that amaurotic family idiocy is due chiefly

to defective vitality of the gray matter of the central nervous system. Sachs is very emphatic in his opinion that the disease is not an acquired one, whereas Hirsch is equally emphatic that it is acquired and due to toxemia. He advocates artificial feeding in these families, while Sachs has shown in some of his patients that the subjects were not breast-fed, and yet were affected with the disease. Poynton and Parsons think the disease is not due either to arrested development or bacterial toxins, but is caused by an inherent biochemical property of the protoplasm of the cells resulting in degeneration of the neurons. They are also of the opinion that the primary changes are in the ganglian cells and the secondary changes in the white fibres.

After a rather careful survey of the literature on the subject, and reading the views of many writers, I am inclined to agree with the view of Cohen and Dixon (1907), in a recent article on this disease, expressed as follows: " It seems to me that the arrested development theory of Sachs, the degenerative theory of Kingdon and Russell, and the toxin theory of Hirsch fit together very well. There is every reason to believe that a child can be born normal and for a short time give no indication that it will not continue in this condition. We have all seen this over and over again; in fact, it is the rule. A child does not cease to develop when it is able to breathe, and the central nervous system is the most backward of all. If the central nervous system fails to develop properly, the finer degeneration must follow, and it is only a step further to the development of toxins due to errors in metabolism." J. H. McKee (1905) also has remarked that: " possibly nutritive or metabolic factors may account for both the arrested development and the degeneration occurring in this disease." As to the biochemical changes spoken of by Poynton and Parsons, I am unable to understand the same, unless they are due to metabolic disturbances.

Differential Diagnosis.—There are other brain diseases of childhood somewhat allied to amaurotic family idiocy, between which it is sometimes difficult to make a diagnosis.

1. This disease is often mistaken for meningitis, but a careful examination of the fundus shows the difference at once, especially after the third or fourth month of age. The condition

within the eyes, together with the other symptoms in amaurotic family idiocy, serve to clear up the diagnosis.

2. Ordinary congenital idiocy is easily differentiated from this special form of idiocy by the absence, as a rule, of the visual symptoms; there is a normal fundus, and life continues for a number of years.

3. Cerebral diplegia. Usually the child is not normal, and there is no special change at the maculæ. Sachs says: "the majority of cases of cerebral diplegia are due to traumation during delivery; the hereditary and family diplegias are more likely to be due to defective development of a considerable portion of both motor tracts. From this point of view, the family form of spastic diplegia may claim to be an allied disease." He says further: "In our classification, we are still hampered by attempts to classify hereditary diseases according to the involvement of the brain or of the spinal cord; but if we drop this narrow, though important, line of classification and if we substitute a broader one including those forms in which there is a defective development of some part of the cerebral, or cerebrospinal system, we can not deny the relation between this form we have been describing and the cases of Friedreich's disease, or cerebellar ataxia, or hereditary spastic paralysis, etc. The difference in the clinical symptoms depends, after all, upon the extent of the developmental defect; whether that developmental defect be most marked in the brain or in the spinal cord, in the pyramidal tracts, in the posterior columns, or in the cerebellar strands, does not matter quite as much as one would at first sight suppose. The underlying causes leading to the development of these various forms of hereditary diseases, are still unknown."

Treatment.—Treatment is of no avail; the disease uniformly ends in death at about the age of two years.

Histologic Report of the Eye.

The eye-ball was removed by Dr. A. A. Hayden, Interne of the Ophthalmologic Division of the New York Post-Graduate Hospital, nine hours after death and placed in Orth's solution. It was received by me two days later. At this time the globe was very soft. Dr. Hayden informed me that this was its condition when enucleated. After leaving Orth's solution, the eye

was hardened in alcohol of increasing strengths and embedded in celloidin. Sections were stained with hematoxylin-eosin, Van Giesen's triple stain, Weigert's stain for medullated nerve fibres, and by several methods for Nissl's bodies.

On microscopic examination the cornea, iris, ciliary body, lens, vitreous, choroid and vascular system of the eye appeared to be normal. In the optic nerve the connective tissue composing the septa was somewhat increased in amount, indicating a moderate degree of atrophy. The temporal half of the papilla was sharply depressed below the level of the nasal half. Weigert's stain demonstrated an early stage of optic atrophy.

The choroid and *retina* were slightly adherent to each other. The cohesion was noninflammatory in character and may be attributed to coagulation of the albuminous products of post-mortem degeneration. In the macular region, the retina was thickened and thrown into folds. The fovea centralis formed a sharply-pointed, papilla-like detachment, projecting 0.75 mm. into the vitreous cavity. The space beneath this detached portion contained freshly-broken-off sections from the cone layer, rod-shaped pigment granules from the pigmented epithelium, and a granular coagulum. These retinal changes cannot be considered pathological, as the formation of folds around the macula are well-recognized as a post-mortem product. The inner segments of the *rods and cones* were well preserved. The outer segments had suffered the usual post-mortem degeneration. The *external limiting and external granular layers* showed nothing abnormal. The *external reticular layer*, which has frequently been described as edematous in this disease, showed less separation of its fibres than is usually observed in microscopic examination of healthy eyes. Near the fovea, the *internal granular* layer appeared to be unusually thick; otherwise, no change was noted. The *inner reticular layer* was normal. At the border of the fovea the ganglion cells were massed to a depth of twelve or more layers. In normal eyes the ganglion layer forms a stratum of single cells throughout the retina, except at the macula, where they are greatly increased in number (up to nine layers). Here the inner granular layer also is doubled in thickness. Near the fovea centralis all retinal layers fuse, the cone visual cells almost alone persisting. We must, therefore, attribute the apparent multiplication of cells found

in the ganglionic and inner granular layers to back-folding of these naturally thick areas by the abrupt elevation which has occurred at the fovea.*

The ganglion cells did not appear enlarged. Nucleus, nucleolus and cell reticulum were fairly well shown. The nucleus was usually eccentric. Many cells were surrounded by clear spaces. All efforts to stain for Nissl bodies failed. Probably they were not present, although Orth's fluid is not the proper fixative when they are to be sought for. In many of the ganglion cells, Weigert's stain revealed the presence of black granules in the cytoplasm, which have been so frequently found in this disease.

Although most of the changes here noted are due to post-mortem degeneration or unequal shrinking of the retina from the hardening process, many of them have formerly been looked upon as related to this disease. In all cases that have been subjected to autopsy, extensive degeneration of the ganglion cells in the brain and cord have been found. This naturally leads one to expect similar degenerations in the cerebral layer of the retina. That this is actually the case, has been conclusively proved by Dr. Ward A. Holden, who, in a typical case of this disease, found marked degeneration in the ganglion cells of the retina with entire absence of Nissl's bodies. The successful demonstration of these minute changes requires fresh material and special methods of preparation. It is to be regretted that these conditions were not fulfilled in the present case. I submitted my slides to Dr. Holden, who kindly examined them, but he would add nothing to the above findings.

This examination proves that the characteristic ophthalmoscopic picture found in this disease does not depend upon retinal edema. It is highly probable that, as in obstruction of the central artery, the appearance results from degeneration and loss of transparency in the ganglion cell and nerve fibre layers.

The microscopic findings which tend to confirm the clinical diagnosis are:

1. The total absence of all inflammatory processes or other

*In some reports, multiplication of ganglion cells at the macula is described as a pathological process. Pathological proliferation of ganglion cells in the central nervous system has not yet been established.

lesions which explain the ophthalmoscopic picture or blindness;

2. Presence of peculiar granules in the ganglion cells.

3. Commencing optic atrophy.

All efforts to produce characteristic staining of the tissues by Nissls' method failed. The absence of the bodies is, therefore, only negative proof.

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A CONTRIBUTION TO THE STUDY OF THE CALMETTE OPHTHALMO-TUBERCULIN REACTION, BASED UPON A STUDY OF FORTY CASES.

BY CHARLES OGILVY, M.D.
Adjunct Professor of Orthopedic Surgery, New York Post-Graduate Medical School and Hospital.

The recent observations of Calmette and Wolf-Eisner on the conjunctival reaction to tuberculin in cases of tuberculosis, have attracted universal attention. This is but a natural sequence; for, when we consider the world-wide ravages of this disease, it is not surprising that any additional light which may be shed upon this subject should be received with worthy credit and widespread interest.

This method of diagnosis was suggested by Wolf-Eisner at a meeting of the Berlin Medical Society, when the subject of von Pirquet's tuberculo-cutaneous reaction was under discussion. About the same time (June, 1907) Calmette, of Lille, France, described the method and reported upon the reactions which had followed the administration of the instillations in 25 cases. Sufficient time has now elapsed to enable other observers to report upon the subject. From these combined reports we are able to form an opinion of the value of this means of diagnosis.

The cases tabulated below have been chosen with the object of determining the efficiency of this method of diagnosis, especially in cases of bone and joint tuberculosis. They comprise the largest series of such cases thus far reported. The work was conducted in great part in the Post-Graduate Hospital.

Preparation of Tuberculin Used.—The tuberculin preparation employed by Calmette is a one per cent. (1%) aqueous solution of the precipitate obtained on the addition of alcohol to tuberculin. We are indebted to Dr. William H. Park, of the New York Health Department, for a supply of this preparation. "A quantity of Koch's old tuberculin (concentrated) is poured into two volumes of 95% alcohol, allowed to settle and filtered through paper. The sediment is washed with 70% alcohol, until the filtrate runs clear. It is then pressed between layers of filter paper, to remove excess of moisture, scraped into a dish, dried in vacuo over sulphuric acid, and triturated in a mor-

tar. Solutions of the powder are made in sterile normal salt solution of 1% by weight, boiled in a water-bath, filtered, and diluted as required." The solution is then put up in the quantities desired, these receptacles being sealed and boiled ten minutes.

The reliability of the test depends largely upon the preparation used, and the technic employed.

Technic.—The importance of proper technic has not been sufficiently emphasized. The patient should lie down and the conjunctivæ of both eyes be carefully examined and any differences noted. The measured drop of tuberculin should then be instilled into one eye, with a finely-drawn pipette held parallel to the eye. The lower lid is drawn well outward and downward away from the eyeball, when the tuberculin is dropped into the bottom of the conjunctival sac. Special care should be taken that no part of the drop is retained on the edge of the eyelid or upon the eyelash; as may result from a sudden movement of the eyelid or head of the patient. Should the patient cry, the test is of little value. The subject may not actually shed tears, but there may be sufficient lachrymation to half fill the conjunctival cul-de-sac; if the drop is then instilled it may float upon the surface of the secretion and, when the traction on the lid is released, be expelled to the eyelash, whence it is wiped off a few minutes later by the patient. It is, therefore, essential to have as little lachrymation as possible.

The reaction has been fully described by several writers. A brief description here of the changes taking place will, therefore, suffice. As a rule, between the third and eighth hour after instillation, a slight swelling appears and a diffuse redness is noticed spreading over the inferior tarsal conjunctiva. These changes are accompanied by slightly increased lachrymation and s metimes by a sense of irritation and burning. The congestion increases, extending upwards over the ocular conjunctiva. The redness deepens in color, and fine whitish, fibrinous filaments are seen on the eyeball and palpebral conjunctiva. Lachrymation is now much increased. In the more severe form of reaction these symptoms are exaggerated. There is slight photophobia, abundant mucopurulent secretion, and tenderness on pressure, marked swelling of the lids and a more generally diffuse conjunctival involvement. The most intense

reactionary symptoms usually develop in from eight to eighteen hours. The cornea, iris and deeper tissues are apparently not affected, nor has the writer noted any pupillary changes. The acute stage of the reaction usually passes off within a few hours, and the eye returns to normal in from one to four days. In some cases a mild conjunctivitis may remain for from two to three weeks.

An attempt has been made to classify the different forms of reaction under several divisions, relative to the degree of intensity of the inflammatory changes. In any such classification, simplicity is much desired, and I would suggest that, if there is to be any division, it should be into two classes only, viz., mild (+) and severe (++). Any further division is not practical and, furthermore, there is no definite evidence of any relation between the intensity of the reaction and the activity or the extent of tubercular involvement.

Untoward Effects.—Several observers have reported unfavorable results, such as long-continued conjunctivitis, keratitis, temporary loss of vision, and ulceration. The contraindications are inflammatory affections of the eye and eyelids—conjunctivitis, blepharitis, keratitis, iritis, and trachoma. If these are carefully avoided and the preparation used has been properly prepared and a small drop used, there should be no apprehension of any unfavorable sequelæ.

CASES.

Name.	Age.	Diagnosis.	Reaction.	Remarks.
1. A. R.....	3	Pott's Disease (3d dorsal)	+	
2. A. B.....	7	Pott's Disease (high dorsal)	+	
3. F. R.....	13	Pott's Disease	+	
4. A. W.....	3½	Rt. hip (with discharging sinuses)	+	
5. E. S.....	3½	Pott's Disease (12th dorsal)	+	Three instillations. After 1st instillation there was no reaction; the child cried at the time of instillation. A decided rection followed the 2d and 3d instillations, which were given four weeks apart.

Name.	Age.	Diagnosis.	Reaction.	Remarks.
6. G. W.....	5	Pott's Disease (12th(dorsal)	+	
7. J. Y.....	13	Pott's Disease (1st and 2d lumbar)	++	
8. P. S.....	3	Pott's Disease	+	Two instillations. First "doubtful."
9. C. S.....	4	Pott's Disease (6th and 7th dorsal)	+	Two instillations. Reaction delayed in both instances.
10. S. L.....	11	Pott's Disease (5th and 6th dorsal)	+	
11. J. N.....	11	Tub. L. knee (discharging sinuses).	++	
12. A. G.....	7	Hip disease (Rt.).	+	
13. J. C.....	15	Pott's Disease 4th and 5th dorsal)	++	
14. C. McD...	6	Hip disease (Lft.)	+	Slight conjunctivitis for 3 weeks.
15. J. K.....	2½	Tub. dactylitis	+	
16. L. A.....		Tub. elbow	++	
17. T. H.....	3	Pott's Disease (with abscess).	+	
18. M. S.....	4	Double hip disease	—	Two instillations. (See conclusions below).
19. L. S.....	3	Hip disease (Rt.)	+	
20. R. C.....	3	Pott's Disease (cervical).	+	First instillation negative; 2d instillation 11 days later. Reaction limited to lower lid below the pupil, with a definite line of demarcation.
21. S. H.....	6	Hip disease (Rt.)	+	
22. W. B.....	7	Hip disease (Rt.)	++	
23. J. B.....	2½	Tub. ankle.	+	
24. C. H.....	10	Lft. knee (traumatic).	—	Two instillations, both negative. Uunder observation.
25. J. G.....	3	Pott's Disease (1st dorsal).	+	
26. M. H.....	9	Hip disease (Rt.)	+	
27. M. S.....	12	Hip disease (Rt.)	+	
28. S. Z.....	7	Pott's Disease	+	Two instillations; 1st negative.

Name.	Age.	Diagnosis.	Reaction.	Remarks.
29. L. S.....	10	Traumatic inflam. left knee.	—	Injury two weeks previous.
30. D. C.....	5	Pott's Disease	+	Two instillations, first negative.
31. G. S.....	3½	Coxa vara.	—	
32. J. L.....	2	Tub. glands of neck.	+	
33. E. L.....	3	Acute osteomyelitis (tibia).	—	
34. J. C.....	3½	Rickets.	—	
35. R. M.....	3	Rickets.	—	
36. J. P.....	3	Rickets.	—	
37. L. C.....	7	Tub. glands of neck.	+	
38. C. F.....	3	Anterior poliomyelitis.	+	No evidence of tuberculosis Under observation.
39. M. M.....	2½	Bronchitis.	—	
40. S. S.....	3	Empyema (staphylococci)	—	

It will be noticed that no doubtful reactions have been recorded in the above series. All such are practically negative, and have been so considered. The earliest sign of reaction occurred in one case within two hours after the instillation. The latest developed 24 hours after the instillation. The test was tried a second time in this case, when the reaction was delayed 20 hours. In one case all signs of reaction had disappeared within 24 hours; in another case a mild conjunctivitis remained for three weeks.

There appears to be no relation between the intensity of the reaction and the extent of involvement or acuteness of the disease. For example, one case of acute Pott's disease on two different occasions reacted but mildly, whereas other cases without any acute symptoms gave a very severe reaction.

In all instances in which a negative or doubtful reaction followed the first instillation, and a positive reaction resulted from the second instillation, the patient had cried the first time, which probably accounted for the absence of reaction. When a second instillation is deemed necessary, it should not be given in the same eye. In case 18, of double hip disease, in which the reaction was twice negative, the patient was in a

very low condition and had but little resisting power. Death occurred a few days later, from which it may reasonably be inferred that the reactionary powers were at too low an ebb to respond to the test.

Some investigators explain the negative reaction reported in cases known to be tubercular, as due to a condition of low resistance—low opsonic index—as occurs in the negative phase. This, the writer believes to be the case in such instances as the one just cited; but, in his opinion, the reactive powers, or the opsonic index, must be very low indeed to account for a negative reaction in cases of tubercular bone and joint disease.

It is very important to make a thorough examination of the patient in order to eliminate sources of tubercular infection other than that of the bone or joint in question.

I wish to acknowledge my indebtedness to Dr. T. Homer Coffin for his continued assistance and coöperation in this work, without which many of the notes taken on the cases above cited would have been unavailable. I take this opportunity also to thank Drs. H. L. Taylor, W. O. Plimpton and H. D. Chapin, from whose services at the Post-Graduate Hospital a number of these cases have been taken.

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WATER DRINKING IN THE TREATMENT OF CHRONIC CONSTIPATION.

BY SAMUEL G. GANT, M.D. LL.D.

Professor of Diseases of the Rectum, New York Post-Graduate Medical School and Hospital.

Water consumed at appropriate times in reasonable amounts, and at a suitable temperature does a great deal in preventing and curing constipation. I would rather discontinue the use of drugs in the treatment of costiveness than prohibit water-drinking by this class of sufferers. Medicine will unquestionably secure an evacuation more promptly in many instances than will water, but, on the contrary, water will bring about a permanent cure more quickly and do less harm than drugs.

Water, being a physiologic agent, facilitates metabolism and helps maintain the body equilibrium; while purgatives act as foreign substances and are not so well tolerated. Water increases the fluids of the body and is carried to remote sections to cleanse the cells and fibres; at the same time it aids in the elimination of waste material and noxious substances. The drinking of water serves to cleanse the interior structure of the body, while bathing does the same for the outer parts.

Water is healthful, but persons often drink too much or at too *high* or too low a temperature. In the treatment of constipation, indiscriminate water-drinking should be discountenanced. Consequently, when this therapeutic agent is employed it should be with a definite object in view.

Except in special instances, water should never be ingested at a temperature below 50° F.* or above 150° F., because, when frequently taken at these extreme temperatures, positive harm is done to the gastrointestinal mechanism. The copious ingestion of water—the consumption of from twenty to fifty glasses of water daily by persons who believe the more they drink the better will be the result—must be condemned. It is rarely necessary to drink more than from ten to fifteen glasses of water during the day, and usually from five to six are ample. The effect upon the stomach and intestine is much better when it is taken in small quantity (half a glass) at frequent

* Unless otherwise stated, all temperatures Fahrenheit.

intervals (one or two hours) than when the same amount of water is consumed at two or three draughts.

Water is but slightly absorbed in the stomach; consequently, when taken in considerable quantities it is retained and interferes with digestion, distends the stomach, and, by its weight, causes sagging of this organ, favoring gastroptosis and enteroptosis—conditions which aggravate the constipated state. Ordinarily, constipated subjects should drink from one glass to a glass and a half of water on arising, and lesser amounts at short intervals during the remainder of the day.

The pernicious habit of drinking several glasses of iced-water at meal time is very detrimental to health, because the water washes the food down before it is properly masticated, dilutes the stomach contents, interferes with the circulation, diminishes gastric secretion, and in other ways interferes with the digestive process. This eventually leads to indigestion, dilatation of the stomach, constipation, and a host of other ailments. On the other hand, drinking of cool or cold water in moderation has a healthy influence upon the alimentary tract and other organs.

Drinking of cold water is contraindicated in colic; in the treatment of very old and feeble persons; in gastric ulcer, arteriosclerosis, persistent anemia, diseases of the kidney, and in persons who are fatigued or overheated from any cause. Cool water produces a general tonic effect and stimulates the intestinal musculature and mucous glands to greater activity.

Ingestion of warm water has an opposite effect. It soothes and quiets the alimentary tract, relieves irritation and pain, and diminishes or arrests enterospasm.

Copious water-drinking favors the elimination of ptomains and poisons resulting from putrefying food and other causes; it softens the feces and in various ways improves the condition of patients suffering from constipation.

The beneficient effect of water in constipated subjects is due as much, if not more, to its thermic as to its mechanical and solvent action. In persons suffering from obstinate constipation I have witnessed many cures following judicious water-drinking, and attention to diet and hygiene of the bowel, together with efforts of the patient to secure an evacuation after the morning meal.

The increased volume of blood following water-drinking is

of short duration. Owing to this fact, it is necessary in cases of chronic constipation complicated by autointoxication, to renew the water supply as often and in such amounts as may be indicated in the individual case.

Under ordinary circumstances, where water is drunk to relieve or cure constipation, the temperature should be about 70° in mild, and from 60° to 65° in obstinate cases. It should never be ingested at a temperature below 50°, except when the sluggishness of the bowel is complicated by fever or in hypopepsia, etc., or in amounts more than a third of a glass every few hours.

When chronic biliousness and autointoxication are present, a glass and a half of cold water, taken in the morning on an empty stomach, stimulates the stomach, liver and gastro-intestinal tract, and in this way helps to regulate the stools and eliminate toxins from the bowel.

Drinking of *warm* and *moderately hot* water should be discouraged in persons afflicted with chronic habitual (atonic) costiveness, for the reason that a lethargic state and infrequent evacuations are induced through its sedative and relaxing action. Water at a neutral temperature of 96° to 100° acts the same way, but to a less marked degree. Very hot water (125° to 140°), stimulates peristalsis, but, owing to its harmful effect upon the stomach, it should not be employed except in cases of constipation complicated by hypopepsia, gastrosuccorrhea, and gastralgia; and then only when swallowed quickly and in small quantities.

From what has been said, it may be inferred that the temperature of water should be selected for constipated subjects with the greatest care, in order that the full effects of this hydriatic procedure may be obtained in the treatment of the various types of costiveness. To emphasize this fact and to recapitulate, it may be stated that *drinking of cold water* is advisable in *chronic atonic constipation*, for the reason that it stimulates the local and general circulation; improves metabolism and assimilation; favors elimination of toxins of all kinds through its effect upon the skin, liver and kidneys; increases peristaltic action; renders the nervous mechanism more responsive; causes a more abundant secretion of mucus for lubricating the bowel; softens the feces, preventing fecal impaction; augments gastrointestinal digestion, and in other ways

assists in toning-up and improving the condition of the body, especially the gastrointestinal tract; so that the stools become normal as regard quantity and frequency.

The above beneficial results following judicious drinking of cold water are due principally to the effect of the low temperature of the water upon the mucuos membrane, which induces a powerful energizing reaction. Cold drinks produce contraction of the intestinal musculature and not infrequently colic; because of this tendency, drinking of cold water should be prohibited in persons suffering from enterospasm and intestinal obstruction.

In *spastic* constipation, the fecal current is partially or completely blocked, because of an occlusion due to simultaneous contraction of both the longitudinal and circular muscular fibres. Such enterospasms may occur at short or long intervals and last for several minutes, hours, or days.

Hot water (110° to 120°) drinking has almost a specific action in relieving and preventing constipation of this type, for the reason that the high temperature of the water has a decidedly soothing effect upon the bowel; in consequence of which irritability is diminished and the contractions become less severe and frequent, or entirely disappear. In many instances this condition can be relieved more quickly when the water-drinking is reenforced by hot abdominal fomentations, which act in a similar manner. In addition to its sedative action, hot water taken internally is serviceable also in the treatment of *spastic* and *mechanical* constipation, for the reason that the additional water ingested liquefies the feces and softens any scybala which may be present, and helps to carry away mucoid collections.

Several cases have been recorded in which mechanical obstruction has been relieved by copious drinking of hot water. The good results obtained in this class of sufferers have been attributed to both the soothing effect of the water in reducing intestinal irritability and muscular spasms, and its mechanical action in dislodging the cause of the obstruction.

ABDOMINAL RELAXATION.

BY ROBERT HURTIN HALSEY, A.M., M.D.
Adjunct Professor of Medicine, New York Post-Graduate Medical School and Hospital.

If the abdomen of a healthy child in the standing position be watched, we notice that it rises and falls during respiration synchronously with the thorax. Following the inspiratory excursion, there is a subsequent return to the previous contour. There is continual relaxation and contraction of the abdominal wall as part of the respiratory act. The expansion is not in any one plane, but in all directions, just as an elastic bag expands on inflation. The ribs, if we notice carefully, will be seen to be elevated and move outward, laterally as well as forward. An adult, man or nulliparous woman, in socalled normal condition, will show practically the same movements excepting the modification due to artificial conditions. The woman, probably, has more motion present in the thorax, while the man has more in the abdomen. These two types, thoracic and abdominal, are usually to be observed, though there are all grades between. Just as there is tone to the extensor muscles of the arm when the flexor muscles contract, so there is a certain tone to the abdominal muscles which permits them involuntarily to give or stretch before distending pressure, while they contract when that is removed. This distention, however, is brought about by increasing intra-abdominal pressure in excess of the pressure the atmosphere exerts upon the surface. The muscles of the abdominal wall may still retain their tone though they have been stretched, as may be observed after filling the stomach, enlarging uterus, etc. There is a limit, however, to the amount of stretching which may be endured without permanent injury. The effects of excessive distention may be seen in cases of extreme relaxation.

According to the physiologist, inspiration is the result of a muscular action; while expiration is merely the recoil of structures, (lung tissue, cartilage and weight of displaced wall) previously put on the stretch. The return of the abdomen to the position of rest must follow from the action, either of the muscles of the abdominal wall, or of the atmospheric pressure exerted

upon the external surface, or from the partial action of both forces.

During inspiration the negative pressure created in the pleural cavities varies between 6 and 30 mm. of mercury. During contraction the diaphragm exerts upon the contents of the abdomen an equal amount of pressure, and probably more, since it also raises the costal arch. The abdominal contents act as the fulcrum upon which the diaphragm presses. To render this fulcrum effectual, it is necessary that the abdominal wall retain its tone and the level of fulcrum be not lowered.

In the peritoneal cavity, the intestines and solid viscera are in contact and may move freely upon each other. The various ligaments, which serve to limit the extent of the motion of the viscera, are composed of folds of peritoneum containing the blood vessels, nerves and fibrous tissue. The intestines, whether distended or empty, being below and very mobile, resemble fluid in their supporting power. For purposes of study we may liken the abdominal cavity to an inverted truncated cone with elastic walls, closed at both ends with elastic membranes held in place by two bony rings; the upper—diaphragm and bony ring (costal arch)—has greater mobility than the lower (pelvis and perineum). With this formation in mind, it will be easily understood how pressure exerted on either the end or sides must be transmitted to all the retaining walls, and increases the intra-abdominal pressure. It follows, also, since the cavity is closed, that relaxation or expansion of these elastic walls will create lowered internal pressure or suction. The intestines retained in the elastic walls are free to move in all directions to the full extent of the mesentery, and only when the walls relax sufficiently to permit sinking of their floating level, do they pull upon the mesentery for support. For illustration, they may be likened to a boat tied with a painter to the dock at high tide; there is an infrequent short tug upon the cord only when the boat drifts from side to side. As the water level lowers, the tug is more frequent, gradually becomes continuous and finally is an ever-increasing strain upon the restraining cord. Just so the mesentery and ligaments of the viscera receive an increasing strain as the intra-abdominal tension, or floating level, diminishes. The kidneys, though outside of the cavity, are inside the retaining wall and are subject to the same forces. This can easily be

demonstrated by placing in a pail intestines contained in a cloth bag and placing a kidney between the cloth and the side of the vessel. The kidney will be held in place at the upper level so long as the level of pressure is maintained, but it will at once slip down as the floating level is lowered. So much for the floating support and the results of its diminution. Since the abdominal cavity is closed and traction upon the walls produces negative pressure or suction, which is exerted upon all parts, such change will affect at first those organs which are most loosely retained in position. As the intra-abdominal pressure decreases, the hollow stomach and intestines dilate in response, and because of such dilatation their vermicular action is impaired. Interference with the peristaltic action permits accumulation of gas and stagnation of solid contents. The negative pressure will prevent a proper return of the venous blood and thus produce a continual chronic congestion. The dilatation and congestion will impair both secretion and digestion of food in the stomach and intestines. Stagnation of food all along the whole tract results. Stagnation permits fermentation and bacterial growth and a condition of chronic toxemia. Gradual impairment of nutrition and loss of weight follow, or there is insufficient repair after mild illness, such as grippe, colds, tonsillitis, etc., to which these patients seem peculiarly susceptible, probably because of decrease in physiological ability to resist infection. Loss of weight depends not only upon destruction of fat, subcutaneous and intra-abdominal, but also upon degeneration of muscle tissue. Loss of intra-abdominal fat may be so excessive as to create an internal negative pressure. Rapid or extreme loss of fat is accompanied by general impairment of the muscular and nervous systems. The change of the nervous system is evident in a heightened irritability of mental as well as reflex action. The muscles lose endurance and become soft and flabby. Chronic toxemia does not affect the muscular system or the nervous system alone; all the structures of the body suffer, but the extent of the injury may be greatest and more apparent in the muscular or nervous systems. Negative pressure, therefore, results not only from loss of nutrition but also from sudden removal of positive pressure, as after pregnancy, tumors, ascites.

The subjective symptoms, therefore, may be classed as digestive, nervous and obstructive, due to displacement interfering with the function of an organ.

The digestive disturbances are belching, flatulence, attacks of colic from formation of toxic and irritating products, and diarrhea occurring in attacks, or constipation.

The nervous system is easily thrown out of balance and little happenings irritate, causing more or less complete loss of self-control. The temper or disposition changes and becomes less cheerful in its outlook; even a distinct psychosis develops.

The displaced stomach may retain a fair motor power and have only a mild stagnation, or in severe degrees of displacement even retention may exist. Retention may be intermittent or continuous, and add greatly to the severity of the symptoms. Displacement of the intestines probably always precedes displacement of the stomach and may give most of the distress ascribed erroneously to the stomach.

The right kidney may be displaced downward with or without gastroptosis, and may or may not add distinctive symptoms. An intermittent hydronephrosis may occur, or severe attacks of pain with fever, as described by Dietl. The displacement of the kidney may interfere with the bile-duct sufficiently to cause jaundice, as related by Treves. In a small percentage of cases the left kidney may be displaced and give symptoms, but this is rare.

Displacement of the liver may cause jaundice, severe or only a slight chronic icteroidal hue, due to the chronic hepato-intestinal-toxemia. The pain may be severe and distressing when in the erect position. Congestion due to the displacement and interference with the venous return through the vena cava may affect the heart's action.

Dislocation of the spleen is rare. The dislocated, tender organ may be mistaken for tumors of the right iliac fossa or of the pelvis.

Cardioptosis may be present in severe grades of relaxation when the diaphragm has been pulled down by prolonged absence of support, or direct tension through the round ligament and liver.

In apparently slight as well as severe grades of relaxation there is chronic congestion of the venous vessels in the abdo-

men, with changes due to this condition, such as chronic colitis, appendicitis, and uterine disturbances.

The very marked aortic pulsation, so often mistaken for aneurism, is a frequent occurrence and seems to be another expression of the extensive tissue relaxation.

Depending upon the long existent relaxation, the sympathetic system becomes exquisitely sensitive and the ganglia are easily irritated by pressure either of the clothes or of even a full meal.

Patients with abdominal relaxation may, therefore, complain of a multitude of symptoms and may emphasize disturbances referable to the digestive or nervous systems or to one organ.

Physical examination should be undertaken in a systematic and thorough manner in order to learn how great is the departure from normal of the individual as submitted. Inspection and palpation should be made in both the supine and the erect positions, in order to determine what effect actual changes in attitude have upon the position of organs and abdominal contour. Thus we investigate the muscular, nervous, respiratory, circulatory, digestive and urogenital systems.

The reflexes will usually be increased and there will be lack of concentration of the attention. Heart and lungs are negative except when complications are present. Rarely, the heart will be found to be somewhat below the usual level.

Those suffering from relaxation show on inspection that they are well or poorly nourished. The well nourished have slight bulging in either lumbar region, with flattening of the anterior epigastric and umbilical regions. As these people stand, the lower segment or hypogastric region tends to bulge forward, and there is a narrowing of the inter-lumbar measurement. When pregnancy has been the cause of the condition, the change may be very extensive and the separation of the recti is apparent on the least effort that contracts these muscles. If pregnancy has not been a factor, contraction of the recti may be voluntarily made to draw in this lower region. This condition is common to both men and women who have lost muscle tone as a result of previous deposits of fat.

When the poorly nourished are upon the table, there is a different abdominal picture and the resemblance is more nearly scaphoid. The region immediately below the ribs is drawn in, so that there is a resemblance to the truncated cone with small

end up. When these cases stand, the lower segment may or may not protrude. If they have been pregnant, there is usually some bulging, but in the nullipara very little if any. This class may be represented by both men or women. These are the cases in which nervous symptoms are usually found. They are most difficult to manage, because they must gain weight before relief will be permanent.

The stomach may be found low, and may or may not be dilated. Motor action may or may not be efficient, and the secretion may or may not be altered.

The intestines are low and the colon may be more or less easily made out.

Single or double nephroptosis may be present, with or without adhesions in a low position.

Hepatoptosis may be more or less severe. The spleen may be found in the right iliac fossa or in any position between that and its normal position.

The uterus may or may not be retroverted or prolapsed.

A continual low intra-abdominal pressure in both types is present; in one because of the pull of the pendulous abdominal wall and weakened muscles, and in the other because of insufficient internal abdominal contents—too little fat—insufficient because the skeleton rings (pelvis and costal arch), maintained at a fixed distance by the spinal column, provide a larger intra-abdominal capacity than the contents require. Since it is a closed cavity, there is a constant tendency to create a negative pressure.

The etiology, therefore, of the condition can be grouped under the headings:

1. Congenital defects.
2. Pregnancy.
3. Prolonged subnutrition.

Under congenital defects should be grouped, probably, all cases found early in life and not dependent upon the action of the other two.

Repeated pregnancies at short intervals, before muscle repair is possible, will leave not only the abdominal wall but also the pelvic floor relaxed.

Subnutrition may result from unhygienic modes of life willingly followed, as among the well-to-do; from close-confining, prolonged labor in factories, as among the working classes.

Corsets, falls and direct blows in the regions of the various organs, probably have no effect on their position unless one of the previously mentioned causes has first existed.

When chronic toxemia has become established, a vicious circle at once becomes active. The nervous disturbances affect digestion deleteriously, while the stagnation in the digestive tract increases the poisoning of the circulation and so hastens degeneration of the nervous system and muscles. Each condition reacts upon the other to exaggerate it.

There are no characteristic changes in the blood or urine. The blood may show secondary anemia; the urine may contain some albumin and casts, with indicanuria, and decreased sulphates and urea-uric-acid ratios.

For purposes of management, the cases may be grouped according to symptoms.

1. Those in whom the nervous symptoms are most prominent.
2. Those who complain most of digestive disturbances.
3. Those in whom organic displacements account for distress.

The aims of treatment must be to relieve toxemia, improve nutrition, replace dislocated organs.

Treatment may be grouped under the means at our disposal, as hygiene, rest, exercise—passive and active—mechanical support, drugs and operation.

Hygiene includes regular hours for sleep, bathing, eating and life in the open air and is necessary for every case. The clothing about the body should be worn loose. The custom of wearing about the waist tightly-constricting bands, belts or strings from which the skirts or trousers are supported, must be discontinued. All weight of the clothing should come upon the shoulders; otherwise, the good arising from support of the lower segment will be counteracted by the downward-pull of the constricted upper segment.

Rest may require prolonged application in isolation, as for simple neurasthenia.

The diet should consist of all classes of food taken at frequent intervals in small amounts. Rapid improvement of nutrition follows the use of such foods properly prepared as to be most easily absorbed from the weakened digestive apparatus. Fluid should be used in large amounts and administered at such times as not to interfere with food digestion. Rectal enemas of

physiological salt solution may be necessary to cleanse the bowel as well as to increase fluid absorption.

Exercise should comprise at first passive massage and resistent motions; later the active use of all muscles. Particular attention should be directed to increased functional use of the abdominal muscles by the exercises. The exercises should preferably be enjoyable and in the open air. Under no circumstances should exercise be carried to the point of fatigue, though a brief rest after each period of exercise will accelerate the gain.

Mechanical support is directed to applying pressure to that segment of the abdomen below the umbilicus. While in no cases do radiographs show replacement of hollow viscera, yet effectual support of the lower segment does relieve the symptoms and aids materially in improving the nutrition of ambulant cases. Improvement of nutrition may accomplish only the relief of the symptoms, yet in some cases it effects complete fixation, when the mechanical support can be dispensed with.

Mechanical support, except in separation or excessive relaxation of the abdominal muscles, should be employed only as a temporary expedient until the above four means of improvement can accomplish their aim.

The mechanical method selected is of little moment, but it is absolutely essential that the lower segment be held back; otherwise, there will be no benefit. It may be noted here that the application of this support relieves symptoms even when there is no prominence of the lower segment, and without raising the lower border of the stomach. The pressure exerted probably diminishes both the strain on the ligaments and the chronic congestion, and in that way relieves the symptoms.

Such drugs should be employed as will improve the tone and meet the symptoms at the moment. In nervous cases, it may be necessary for a while to give sedatives or hypnotics, but they should be used only as a temporary expedient and never for prolonged periods. Glycero-phosphates, iron, arsenic, ergot, cocoa, strychnin, may all be employed. Cathartics, however, should be employed only on rare occasions, depending upon the tonics and irrigations to empty the bowel.

When there are definite symptoms from displacement of an organ, operation must be resorted to. The pelvic organs

should be replaced and repaired, if there are lacerations or prolapse. The kidney should be fixed, if it has an intermittent blocking of the outlet. The liver should be sutured in place, if it is causing pain because of its low and moveable position. A dislocated spleen may require surgical operation to replace and fix it.

Too great emphasis can not be given to the need of careful study of each case of abdominal relaxation to determine its cause, and then the exercise of judgment and perseverance in the application of the principles indicated in this brief outline.

REMARKS ON CICATRICIAL STRICTURE OF THE ESOPHAGUS, WITH REPORT OF A CASE.

BY EDWARD W. PETERSON, M.D.
Adjunct Professor of Surgery, New York Post-Graduate Medical School and Hospital.

William H., aged 22 years, a plumber, was employed during the summer of 1905 as a "Spieler" for a Coney Island show. He developed "speaker's" sore throat, for which condition he was taking a cough-mixture. One day he swallowed a corrosive acid by mistake, instead of the cough-mixture. He has kindly written an account of the accident, which I submit in his own words:

"On the 10th of July, 1905, I drank nitric acid by accident. I felt very intense pain in my mouth, also in the esophagus. About forty seconds after I drank the acid, I was given a large glass of milk to drink, and a few minutes later I was taken to the Emergency Hospital of the Park. There they gave me some kind of stuff to cause me to vomit, and magnesium sulphate solution to drink. They also washed out my stomach with the same solution I was then transferred to the King's County Hospital. During all this time I had very bad pains in the stomach. I felt weak and very thirsty. My bowels moved very often, which I think was the magnesium sulphate. After my stomach was washed I had pains in swallowing, just as if I had caught cold and had a sore throat, but I was able to swallow just the same, except for the pain I had. I also found it very hard for me to speak.

"That night I could not sleep because it was impossible for me to rest my head; no matter how I laid, my neck pained me terribly. That night I drank a lot of water, but it all came up again. The next morning I still had pains in my throat, but I was able to swallow. I also stopped vomiting the next morning. The pain in my throat kept up for about six days, then gradually went away. For three days and nights I was constantly kept awake by hiccoughs.

"About eight days after I drank the acid, I noticed that while eating meat or so-called hard food, it did not slide down so very easy, and gradually it got so that I was able to eat only soft

food, and after a few weeks it got so that I had to live on fluids, and at times even fluids would not go down."

In the latter part of August this patient came to my clinic at the Post-Graduate Hospital, giving the above history. Several unsuccessful attempts were made with various-sized instruments to pass the stricture, which was located nine inches from the teeth. Finally, a small flexible bougie was introduced, and after a number of days of treatment, a No. 26 instrument could be passed. Following an attempt at divulsion with a metal esophageal divulsor, the patient developed a cellulitis of the neck, and for several days his condition was serious. Fortunately, no ill effects resulted, but the lesson was taken to heart and the divulsion was abandoned. Later the patient went away from the city and for a time was lost sight of. When he returned he was greatly emaciated and unable to swallow water, except a spoonful at a time, and this amount very slowly. All attempts to pass instruments failed. Even filiform bougies could not be introduced. The patient attempted to swallow a perforated No. 4 shot with a thread attached, but without success.

Dunham has demonstrated that when a patient can swallow water, a thread can be washed through the stricture. This fact was taken advantage of, and a thread was washed through the stricture into the stomach of the patient. A temporary gastrostomy was performed, the end of the thread fished out, and to it was attached a strong fish line which was drawn up through the esophagus out of the mouth. With this guide it was easy to draw up wire spindle bougies and, as long as there was no marked resistance, gradually to dilate the stricture. Later, with a full sized instrument in place, an internal esophagotomy was done, using the Abbe string method to chafe through the stricture. Systematic dilatation was kept up, covering a period of several weeks, until a No. 42 flexible bougie could be passed. The patient rapidly regained his strength and increased about fifty pounds in weight. He was taught to introduce the bougie and instructed to pass it once or twice a month. He has been kept under observation now for nearly three years and there has been no recurrence of the stricture.

General remarks.—While esophageal stricture is a comparatively rare condition, it has been my good fortune personally to treat eight cases, as follows: One case of compression stenosis,

due to mediastinal sarcoma; one case of carcinomatous stricture (permanent valvular-gastrostomy was performed, with feeding through the gastric fistula); two cases of foreign body obturation; four cases of benign cicatrical stricture, two of which were due to swallowing caustic acids, and the other two due to ingestion of caustic alkalies. In the summer of 1905, Dr. Samuel Lloyd, while away on his vacation, placed under my care one of his patients who had a diverticulum of the esophagus which caused marked dysphagia. Besides these cases, it was my privilege, when an interne in the Post-Graduate Hospital, to see and study six additional cases of cicatricial esophageal stricture, and personally to carry out many of the details in connection with the treatment.

The diagnosis of this affection is easy. No matter from what cause the stricture arises, there are certain constant symptoms, varying in degree, of course, with the nature and extent of the stenosis. These symptoms are *increasing dysphagia*, *persistent regurgitation*, and *progressive emaciation*. The physical examination of an individual with esophageal stenosis should include the ordinary methods of diagnosis, viz., palpation, percussion, auscultation, etc., and, in addition, the use of bougies, the X-ray and the esophagoscope. The passage of bougies will determine the site of the stricture, also the patency or non-patency of the canal. Begin first with large instruments, and gradually decrease the size until one is passed or until the non-patency of the esophagus is established. The greatest caution and gentleness should be observed in these efforts; absolutely no force should be exerted, for it is very easy to penetrate the wall of the gullet and thereby to set up fatal inflammation. One should not be satisfied with a single examination, but with various-sized instruments, with the patient's head in different positions, repeated attempts should be made to pass the obstruction. By administering a thick bismuth paste and taking a radiograph of the chest and neck, it is possible to obtain accurate knowledge of the location of the stricture and of the calibre and course of the esophagus above.

In selected cases, the esophagoscope should be used. The introduction of this instrument is somewhat difficult and not without danger, but in the hands of an expert it is possible to obtain an exact picture of the esophagus.

The treatment of the condition under consideration is usually difficult, always protracted, and is calculated to tax the patience and the ingenuity of the surgeon. The dangers attendant upon the forcible introduction of bougies, rapid instrumental divulsion, internal esophagotomy with sharp cutting instruments (on the plan of the urethrotome) are too well known to require either emphasis or repetition. The treatment which is recommended is as follows:

1st. In cases where cicatrization is not extensive, and where the stricture is yielding, the systematic passage of ordinary flexible bougies and the hollow-guide bougies will suffice to keep the esophagus patent and eventually overcome the tendency of the scar to contract.

2nd. Pervious strictures which do not yield readily to the ordinary divulsion methods should be treated by internal esophagotomy. This is best accomplished by the Dunham string esophagotome. The guide-bougie is passed through the stricture, and an olive larger than the stricture is engaged. By a sawing movement, the string chafes through the stricture without danger of hemorrhage. This procedure is to be repeated after a few days, until the lumen of the esophagus is of a satisfactory calibre.

3rd. In cases where the introduction of an instrument through the stricture is impossible—if the patient can swallow water—it is possible to wash a thread through the stricture into the stomach. An ordinary drinking-tube, a thread, and a glass of water are all that are necessary to accomplish this result. A gastrostomy (temporary) should be performed and the thread drawn out through the gastrostomy wound. A double linen fish line should be attached to the thread and drawn up through the esophagus into the mouth. With one of these lines, the wire spindle bougies of various sizes, should be drawn up, beginning with the smallest, until a spindle is arrested. Pulling the wire taut, thus putting the stricture on the stretch, the other fish line is pulled to and fro, until the stricture is chafed through and the spindle is allowed to pass upward into the mouth. The string should be left in place and the operation repeated a few days later until sufficient dilatation has been obtained. The string is then removed and the gastrostomy wound will heal spontaneously.

4th. In impermeable strictures, where all attempts at divulsion have proved unsuccessful, a permanent valvular gastrostomy should be established.

In conclusion, attention should be directed to the following points:

1. Forcible introduction of bougies, rapid instrumental divulsion, internal esophagotomy with sharp cutting instruments, etc., are positively dangerous and should not be employed.

2. Before attempting any dilatation of an esophageal stricture, a guide-bougie or string-guide should be passed.

3. The treatment does not end with the operation. Strictures are prone to relapse; therefore, vigilant supervision must be maintained in each case until such tendency has disappeared.

ON EXCESSIVE GASTRIC SECRETION.

BY L. KAST, M.D.
Instructor in Medicine, New York Post-Graduate Medical School and Hospital.

According to present views, excessive secretion is divided into two forms: hyperchlorhydria and hypersecretion. They are considered as separate diseases. By hyperchlorhydria is understood an abnormal secretion where the gastric juice contains an increased amount of hydrochloric acid in the unit of quantity. By hypersecretion is understood a quantitive change of secretion, there being more juice but containing a normal amount of hydrochloric acid.

Our knowledge concerning the increased secretion of gastric juice is of long standing. Excessive gastric secretion was recognized even when diseases were diagnosticated by subjective symptoms and sometimes by the quality of the vomited matter. The terms hypersecretion and hyperchlorhydria date back, however, to the time when the stomach-tube was used for diagnostic purposes, and especially since systematic examinations of the stomach function have been carried out with Ewald's test-breakfast. There is no question that in the last two decades remarkable progress has been made in gastrology. Diagnosis and treatment have been put on a scientific basis instead of following empirical rules.

These functional examinations, however, have not thus far furnished a satisfactory explanation of these secretory diseases, nor do they establish a reliable separation between each other. Especially the relation between increased gastric juice and other diseases—like peptic ulcer, or chronic catarrh of the stomach, neurasthenia etc.—has not been elucidated, in spite of the enormous accumulation of clinical observations.

A new phase in the understanding of these problems began with the work of Pawlow[1] and his pupils, and with the studies of Cannon[2] on the motility of the stomach. New facts have been brought forward and added to the physiology of secretion and motility. In the last few years the attempt has been made

1. Pawlow: Work of the Digestive Glands, London, 1903.
2. Cannon; *Amer. Journ. of Physiology*, 1907.

to establish, with the newly discovered physiological laws, new explanations (Rubow, Bickel) of diseases of the human stomach. We are approaching a radical evolution of our conception of digestive problems. Many points will have to be corrected and quite a number will have to be modified.

There is a gradual transition from complete absence to continuous secretion of copious, highly peptic gastric juice. Nature does not draw any decided demarcation between the different forms of secretory anomalies, and it is impossible to reach a thorough understanding of them without taking them all into consideration. This is unquestionable for clinical research.

For practical purposes, however, the classification into diminished, and excessive secretion, and their consideration as separate diseases is of great value. In the following, the question of excessive gastric secretion is considered, ignoring all the different aspects of the problem except the point, whether there is sufficient reason to retain the present division into hypersecretion and hyperchlorhydria, as separate diseases. This problem can be attacked from two sides: (1) experimentally; (2) clinically upon the experimental facts. The following remarks deal with the experimental side of the question.

The really pure gastric juice is the product of the oxyntic and principal cells. This juice is a clear fluid and contains 0,4—0,55% HCl (which is equal to 100–150 cc. 1/10 normal hydrochloric solution) and varying amounts of pepsin and rennin. It is very difficult to obtain the pure juice of the stomach glands, because there is always more or less gastric mucus upon the surface of the gastric mucosa, besides the swallowed saliva and mucus of the mouth and throat, which we will not consider. This mucus is alkaline; it binds partially the hydrochloric acid and influences in this way the acidity of the juice. The more gastric mucus is mixed with the specific gastric secretion, the more is this alkalinized.

The less mucus is poured into the stomach, the less the gastric juice is diluted and the nearer it is to the normal acidity. We, therefore, have to make a distinction between the physiologic conception of the gastric juice, *i.e.*, the pure secretion of the specific gastric glands without the gastric mucus, and the clinical conception, *i.e.*, the juice with the gastric mucus. In speaking of the acidity of the gastric juice after a test-breakfast, we have in mind its clinical conception.

If a dog is operated upon according to Pawlow's method and a sham-feeding is given, it is possible to obtain the gastric juice from the fistula. When it is collected from time to time as it pours out from the fistula, it can be shown that the different portions vary with regard to quantity and acidity. Diagram *A* shows this graphically. Portions in the beginning and toward the end of a secretion show less acidity than the intervening portions; this is due to the fact that at the beginning and at the end of the secretion there is more mucus mixed with the juice.

When the gastric secretion is at its height, there is only a small quantity of mucus visible and the acidity of these portions differs from each other very slightly. These facts, together with quantitive analysis of the hydrochloric acid, justify the assumption that the gastric juice has a constant acidity. It is the amount of the admixed mucus which accounts for the difference in the acidity of the different portions during secretion. When the gastric secretion is at its height, the total acidity of the juice is between 130–160. These numbers stand, therefore, for the pure gastric juice of the dog (without mucus). The same conditions hold good for gastric secretion which is not stimulated by psychical influence alone as sham-feeding does, but also by the presence of food-stuffs in the stomach. According to Pawlow, this can be shown on dogs which have a small stomach.

It is easy to show that the amount of gastric juice is very variable depending upon the appetite of the animal, or upon the quantity and quality of the food. The gastric secretory apparatus is, indeed, wonderfully regulated and pours, under normal conditions, just as much juice into the stomach as is necessary, with regard to the digestive work to be done. It is further easy to show that certain chemical bodies, as for instance, alcohol, peptones, meat extracts, pilicarpin, increase the quantity of gastric juice considerably, but it is impossible in the dog to increase the acidity of the juice beyond its physiologic limits.

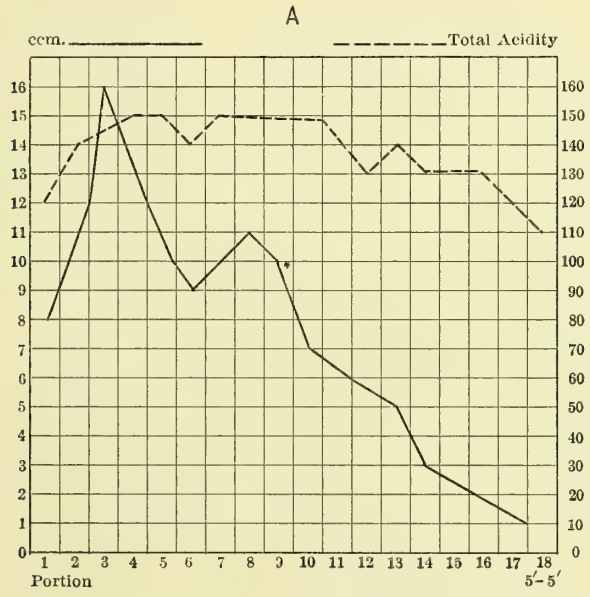
This means that the stomach is prepared to meet the varying demands for digestive work by changing its quantity without altering to any degree the acidity of the different portions.

These facts have been established by Pawlow and have been verified by all competent experimenters. In experiments[3] with

3. Kast: *Arbeiten aus dem Pathologischen Institut*, 1906.

alcohol on Pawlow-dogs, which I carried out in the Pathological Institute of Berlin, I frequently could obtain, after the same food, or after the same sham-feeding, three and four times the amount of gastric juice if alcohol was added or brought into the stomach for a short time before the psychic or local secretory stimuli were applied; but I was unable to modify to any extent the acidity of the juice.

These facts concern the stomach of the dog. The next question is whether these facts correspond with the condition of the human stomach. Recent examinations on human beings, which for

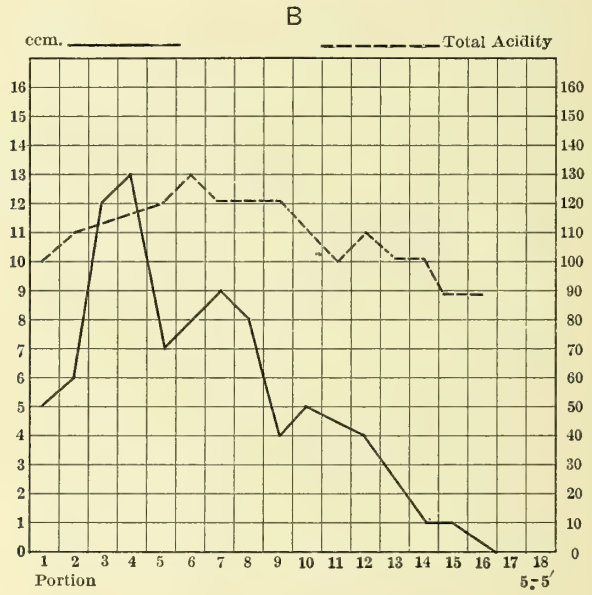


therapeutic purposes have been operated upon in the same way as Pawlow had done with animals, have shown that there is in the principal features a complete uniformity between the human stomach and the stomach of the dog; at least with regard to secretory function. Hornborg, Roeder and Sommerfeld, and especially Bickel[4] have shown that the acidity of the pure gastric juice of the human stomach varies between 0,4—0,55%, (= 100–150cc. of 1/10 normal HCl solution); further, that the psychic and reflex stimuli, duration, etc. are in principle the

4. Bickel: *Arbeiten aus d. Pathol. Instit*, 1906.

same as in the dog. Figure *A* shows graphically the secretion after sham-feeding in the dog. Figure *B* shows the same in the human subject after sham feeding. The similarity between them is apparent.

It was possible also in many other respects to gather points which justify the application of the physiology of secretion in the dog to the human being. Thus, I obtained in the sham-feeding girl, in experimenting[5] with 5% alcohol, the same results as in dogs, *i.e.*, a considerable increase in quantity but no increase in acidity beyond the normal limits.



Most of the experiments in dogs were carried out on healthy animals. It is true that sham feeding necessitates operative preparation of the animal, and anatomically the individual is abnormal, but functionally we have to consider the individual as normal, for reasons which cannot be entered into here. Now, the question is whether the observations obtained on the healthy stomach can be used as a basis for classification and explanation of secretory disturbances so often met in man. I do not know whether there have ever been observed in dogs diseases like

5. Kast: *Archiv. für Verdauungskrankh.* 1906.

hypersecretion or hyperchlorhydria. To attack our problem from this side, it would be necessary to produce these secretory anomalies artificially in animals, and this would have to be done in such a way, as to not represent a temporary alteration, but a chronic state. Experiments in this line have been made only singly and do not allow any conclusions. To explain a pathological process of any organ, it is well to take the element of physiology for the explanation of the pathology, if the studies cannot be made directly in corresponding pathological conditions of the animal. Pathological function is in most cases only a modified or exaggerated physiological function. It might be thus possible in default of corresponding observations on diseased animals, to fall back on observations of functionally normal animals, all the more for the accordance between the man's and the dog's gastric secretion. At least this would be logically more acceptable than the assumption of hitherto unobserved functions.

In order to test a human stomach with regard to its secretion, we stimulate it to this function, and by withdrawing, after a certain time, the stomachs contents and determining the amount of hydrochloric acid, we draw our conclusions. This functional test is generally made by Ewald's test-breakfast. Empirically it has been found that the stomach contents one hour after the test-breakfast show a total acidity of from 40–60. This, of course, does not mean that 40–60 is the acidity of the pure gastric juice. The pure juice after test-breakfast is, for obvious reasons, not obtainable. It means that the gastric juice, after being poured into the stomach and being mixed with the bread and water, the saliva and the mucus from the mouth, throat, and the stomach, assumes this acidity. It is the diluted juice. This dilution is not one gradually increasing during digestion, for the obvious reason that the stomach evacuates its contents in successive portions, during which time new gastric juice is always being secreted.

It will be, perhaps, impossible to give an exact account of the amount of hydrochloric acid present at different times during the hour of the test-breakfast, because a very complicated process of motility, secretion, absorption, and evacuation is encountered. What is learned at the end of an hour by means of a test-breakfast is only a small part of that which precedes it. It is, there-

fore, necessary to bear in mind that conclusions drawn from the acidity of the test-breakfast with regard to the quantity and quality of the secreted juice, have to be taken *cum grano salis.*

Now, if a test-breakfast, instead of showing a total acidity of 40–60, shows one from 80–100 or 120; what does this mean? There are two possibilities:

1. The gastric juice is in this case much more acid, *i.e.* the result of its mixture with the test breakfast, etc. shows a higher amount of hydrochloric acid.

2. The gastric juice has its normal hydrochloric acid, but the amount of juice is increased, or the situation is such that the normally acid juice undergoes less dilution.

The special text-books explain hyperchlorhydria by assuming that a highly acid juice appears under certain conditions. Now, an abnormally high amount of hydrochloric acid for the juice would be, for instance, 200. Such an acidity has never been found even in those cases of continuous secretion in which nearly pure juice can be obtained from the fasting stomach. In a very careful study, Rubow[6] has shown that there is not a single case in the literature where the total acidity has been higher than 100–150, except, of course, in those cases in which the fact has been overlooked that other acids than hydrochloric acid have been found (for instance lactic acid in consequence of retention).

As we have seen, 100–150 represents only the normal acidity. As long as physiology taught that the human gastric juice has an acidity of 50–100, it was justifiable to assume "hyperchlorhydria" by finding acidities of 150. This is obsolete, since it has been shown that physiologists underestimated the normal amount of hydrochloric acid. By finding a total acidity of 100 after a test-breakfast we can, therefore, only say this juice is less diluted than in a normal test-breakfast. This, in turn, is possible when there is at the end of the hour less test-breakfast in the stomach or there is more juice than normally. In short, abnormal motility, or abnormal secretion, or both, can lead to a result which we, by means of a test-breakfast, define as "over acidity."

It is clear that the more acid juice is added to a certain amount of test-breakfast, the higher is the acidity. At the same time it

6. Rubow: *Archiv für Verdauungskrankh.* 1906.

increases the quantity of the mixture. This corresponds to the withdrawal after a test-breakfast of increased quantity with increased acidity.

On the other hand, the more test-breakfast evacuated through the pylorus at the beginning of the hour, the less diluted will be the gastric juice, especially if the secretion is slight in the beginning and copious at the end of the hour. This corresponds to the withdrawal after test-breakfast of increased quantity with increased acidity.

There is another possibility: there may be normal secretion, and at the same time lively evacuation of the test-breakfast throughout the hour. The consequence will be diminished amount with high acidity.

We see that a certain result of test-breakfast can occur with a gastric juice of normal percentage of hydrochloric acid, namely, normal quantity or even diminished quantity with high acidity —which, so far, has been regarded as the basis for the diagnosis hyperchlorhydria.

It is clear in what direction our conception has to be changed. Instead of assuming that the neuroglandular apparatus of the stomach produces, in diseased conditions, a juice of abnormal acidity, (an assumption without sufficient support) we are led to believe that a combination of different functions—extremely changeable—(as quantity of the juice and the motility of the stomach) will account for the variable acidities up to the total amount of 100–150.

We know that there are two sources for the gastric secretion. One is psychic, the other reflex. The pyschic stimuli of the secretion play an important part, but their effect is small in comparison with the stimuli arising from the presence of food in the stomach. It seems to me very probable that the latter stimulus will have to be held responsible as the main etiologic factor of hypersecretion.

Cannon's most interesting studies have elucidated the physiology of gastric motility to a point of completion. Through them it became apparent how closely secretion and motility are linked together.

It is the hydrochloric acid within the stomach which opens the pyloric muscle, and it is the hydrochloric acid which, as soon as it reaches the duodenum, closes the pylorus.

Both effects are reflexes. There is no doubt that the amount

of hydrochloric acid which passes the pylorus will influence the mode in which the stomach empties itself of its contents.

Thus, the acidity in the stomach may become the factor which, under certain conditions, increases its own amount by disturbing the normal pyloric rythm.

Summing up, the following can be said:

The acidity of the gastric juice varies between 100–150.

The gastric juice maintains this acidity, but it is very variable with regard to quantity.

There is no such thing as hyperchlorhydria in the sense that the juice itself becomes abnormally acid.

An increased acidity of the stomach's contents occurs and may reach the height of the acidity of the undiluted gastric juice, but not higher.

Increased acidity is due to hypersecretion, which means an increased quantity of juice, or is due to modified motility of the stomach, or to both factors.

It will be necessary to take these experimentally established facts as a basis for revision of our clinical conceptions. We will have to determine the part which impaired or increased motility and the part which increased secretion play in producing high acidity.

I mentioned above that there are two factors which regulate secretion: a psychic factor, and the presence of certain food-stuffs in the stomach. I also ventured the probability that the latter factor will have to be taken into consideration as an etiologic cause of increased flow of gastric juice. The remarkable discoveries of Starling and Edkins have proven that this part of gastric secretion is stimulated by certain chemical bodies of specific nature. They call them hormones—(messengers). These harmones are formed in the mucus membrane of the pylorus and duodenum and stimulate secretion after circulating in the blood.

From this point of view, it might be possible to explain why the local affections of the pylorus and duodenum are so prone to induce hypersecretion. Many forms of excessive acidity which to-day are called neurosis, will possibly be explained as the effects of abnormal formation of hormones. This would mean that it becomes a problem of internal secretion instead of a " neurosis,"—a frequent excuse for lack of knowledge.

THE HYDRIATRIC MANAGEMENT OF BURNS.

BY FRIEDRICH GROSSE, M.D.
Instructor in Hydrotherapeutics, New York Post-Graduate Medical School and Hospital.

"The treatment here recommended is neither new nor unknown, but is like a rough diamond among a pile of broken glass. It is mentioned in text-books of surgery and resorted to here and there in many variations, but mostly unreasonably, and then with little success. It is misjudged and little valued, and yet it is the first, the most effective, and indeed, the only measure which under all circumstances so powerfully and reliably limits the terrible results of burns. In no other bodily disease do we possess such a convenient, quick, cheap and handy means as in burns, if applied rationally, timely, and continuously."

Thus wrote Dr. Carl Heinrich Dzondi, ordinary professor of medicine and surgery at the University of Halle, Germany, in 1816, in his book on "Burns." He was so enthusiastic that he concluded his introduction in these words: "I implore all humane men, by recommending and distributing this booklet, especially in schools—for it is popularly written—to aid in making this excellent method generally known and applied."

The treatment so ardently advocated is the hydriatric management of burns. The hope and wish of Dzondi have not been realized. The author and his method have fallen into oblivion. Not better than the destiny of Dzondi's book was that of a treatise by Winternitz, in the sixties of the last century. The first paragraph of this booklet reads thus:

"If in our epoch of statistics and proofs one attempts by large figures to bring before the public a subject supported only by relatively little experience, he must have cogent reasons. For instance, surprisingly favorable results, which bid fair to cause a thorough examination of the matter."

In his famous book: "Die Hydrotherapie auf Physiologischer und Klinischer Grundlage," Vienna, 1877–79, which is still and forever will be the foundation of all hydrotherapeutic work, Winternitz refers to his booklet and adds that his hope has not been fulfilled; but in spite of this fact, he feels the necessity of

advocating the hydriatric treatment of burns anew, because it is more effective, more useful and more rational than the methods of treatment now in vogue.

When making hydrotherapy a special study, I read these suggestions of Winternitz and followed them. After an experience of fifteen years I have concluded that the advice of Dzondi and Winternitz is true. Whatever I saw in Germany and the United States could not induce me to abandon the precepts of Winternitz, the father of hydrotherapy. What he and Dzondi taught is still valid; the enormous success of bacteriology and of modern chemical industry with its new drugs without number, cannot invalidate the intuition of these able observers. My own experience and the assurance that it will help to lessen human ailments and counteract the terrible and often fatal results of burns beyond what can be done so far, give me courage once more to advocate the hydriatric management of burns, even though my first effort in the *New Yorker Medizinische Monatsschrift*, for October, 1902, shared the destiny of similar attempts on the part of my famous predecessors. Recent publications and researches and the fact that the men who warn against "gross cleanliness in surgery," increase in number, present fairer prospects of success.

After a review of the literature of burns, and actuated by experience, we may present the following:

Technique, How to Manage Burns.

I. *If a Bath-tub is at Hand.*—If the *whole* surface or a larger part of the skin is burned, rendering a partial bath impossible, the patient is placed in a full bath, the temperature of which should be from 80° to 88° to 93° F. The larger the afflicted area, the warmer, as a rule, the water should be. In the beginning, the temperature may be low, but later it should be 99° to 90°; this is regulated entirely according to the patient's feeling and the requirements of the case. After the accident the clothing should not be removed, except the loose ones, and of course the shoes. As soon as the first shock and the worst pain are over and all preparations for the continuous bath have been made, the patient should be undressed and manipulated as necessary.

If only *a part of the body* has been burned, which permits a

partial bath, only this part is placed in a suitable vessel; again without undressing and without cleansing, except, of course, when caustic chemicals are in consideration. The temperature may be from 70° to 85°F. and less, but extreme degrees should be avoided. The feelings of the patient will be the best indicator. The duration may be from fifteen minutes up to days, especially in necrosis. If the worst pain is relieved, the clothes may be removed and the part cleaned; in so doing, all disinfectants must be avoided and even the cleansing should be done in a somewhat perfunctory manner. Blisters should not be opened. The partial bath may now be continued at a higher temperature, 90° to 99° for a longer time, or the part dressed as detailed later.

II. *If no Bath-tub is at Hand.*—If the *whole* body is affected, a woolen blanket is spread upon a bed, over this rubber tissue, and over this two wet bed sheets, and, finally the patient is lifted upon these; the upper sheet is now cut so that each part of the body can be covered separately by wrapping the strips around the parts, or by gently pressing the pieces smoothly upon the surface. One end of the bedstead is elevated a little, and water of 50° to 70° is poured from time to time without pressure over the aching parts. If the pain ceases, the underlying wet sheets and dry blankets may be closed over the patient to warm him. Cold extremities may be warmed by a warming bag. In case this management is impossible in emergency, the water is simply poured upon the burned undressed patient, or, if unclothed, he is covered with some cloth, to prevent all mechanical irritation. In burns of the first degree, the treatment so far outlined will be sufficient. In burns of the second and third degrees of the whole body, the patient afterwards is placed in the continuous bath as already described.

If only *a part of the body* is burned, this part should be undressed and, without disinfection and cleansing, covered tightly and smoothly with one or two wet layers of fine linen, muslin, or similar material, or two to three wet layers of plain absorbent gauze. According to the locality—for instance, around an extremity—a gauze roller may be wound twice, or these layers may be placed only upon the burned area, where rollers cannot be used. These two primary layers (this phrase will repeatedly be used for the sake of brevity) remain upon the burn for from one to two days. The object is to save

the part from mechanical irritation. Upon this layer cold compresses are applied, wrung out of water at 50° to 70° F., to be removed whenever the pain increases. This means very often in the beginning, sometimes every minute, and subsequently at longer intervals. If a cooling coil is at hand, it may be used with water at a higher temperature, about 65 to 75° F. Extreme temperatures upon the burned area are irrational; so also are ice bags. If the hand and forearm or the lower part of leg are burned, it may be useful to apply ice in any form near the healthy part in addition to the local dressing.

In burns of the first and second degree, this treatment will suffice. Blisters, if not too large, will be absorbed within one or two days, or may be now cleared away. During this time the pain first caused by the burn is entirely gone. In burns of the third degree, pus now begins to appear, and inflammatory pain, with an inflammatory halo around the burned area, may arise. This is the time to remove the "primary layers," as I call them. Pieces of plain absorbent gauze, five to ten layers, according to the quantity of oozing, are now cut in shape and size, so as to cover exactly the burned region. The gauze is wrung out of an antiseptic solution, as detailed subsequently, placed upon the wound and covered with a soft impervious stuff, one inch or so larger than the gauze. The dressing is then fastened by a roller that presses the impervious cover at all parts against the healthy skin, in order to prevent drying of the wet gauze. This dressing has to be removed whenever pain arises or the gauze is saturated with pus. Instead of pervious covering and gauze roller, it may be preferable to apply over the wet medicated gauze a common wet compress (a towel wrung out of plain water) and covered with a woolen cloth or flannel—not with an impervious stuff. This applies, for instance, to burned fingers, where the medicated gauze is applied to each finger separately and the towel over the whole hand.

PHYSIOLOGICAL REASONING.

In burns, our task is a fourfold one. We have:

1. To relieve pain.
2. To combat the fatal results in extensive cases.
3. To prevent the patient from being injured by irrational manipulation; and
4. To cure the results of the burn.

As to pain, there is no doubt but that it can best be relieved by baths and compresses. In the bath we use low temperatures, which, as we know, reduce sensibility. Indeed, the moment the burned part is brought into contact with the water in the bath or the compress, the pain ceases. To illustrate this, I quote the younger Hebra: "The burned, who had filled the house with their screams, become quiet within a few minutes after they have been placed in the water, and they say that they are comparatively well." One of his cases would not believe that his back, which he could not see, was burned as badly as his front, which lay open to his view.

As to the temperature, we have a wide range. Dzondi wrote as early as 1816: "It is not necessary to use a thermometer to obtain a certain degree; it is always sufficient and rational to be guided by the feelings of the patient; but to employ water near or at zero would act rather disadvantagously. The circumstances of the moment and the condition of the patient must guide us." Of course, as we have to act as quickly as possible, we should not lose time to obtain a certain temperature, but, as soon as practicable, we should secure the degree that is in every way as favorable as possible. Local baths may be cooler; full baths will very soon have to be warmed to 88°–93° F. and more, to prevent too pronounced loss of heat. This can be done, the more so as the effect in a full bath is cumulative and the bath is altogether sedative, and offers an ideal rest to the sufferer.

Further, it must be appreciated that we need not irritate the skin by cleaning it or removing the clothes (a procedure extremely painful; for this reason the patient is narcotized, which certainly does not act favorably in regard to general conditions); only the thermic factor is active, and by the primary layer, as I call it, even the slightest movement and irritation through the changing of compresses are avoided. By it we form, so to speak, an artificial epidermis. This mode of relieving pain is certainly superior to morphine, in so far as it is in accordance with the three other indications. Of course, there is no reason why the administration of morphine may not be combined with the water treatment.

Another advantage is that this treatment is adapted also to the second indication in the treatment, that is, to combat the fatal results of extensive burns. Thus far there is no

unanimity regarding the question as to what may cause the severe general condition following such burns and the cause of death. Ponfick and Lesser point to the oligocythemia caused by the destruction of red corpuscles; Tappeiner, to the loss of blood plasma; Silberman taught that the thrombotic processes in the internal organs caused the fatal symptoms. The best explanation, however, seems to be that of Sonnenburg. He found, by experiment, that death occurs through over-excitation of the nervous system and by reflex relaxation of the tone of the vessel walls, and, besides, the overheated blood may cause cardiac paralysis. However this may be, there is hardly any doubt that from what we know to-day of the physiological effect of the continuous bath, it can act only favorably. Hebra, senior, was long thought to have been the first to apply the continuous bath in burns, until A. Rose dug out of the older literature that Passavant, surgeon of the hospital in Frankfurt a/M., Germany, had used it before, and had written an able essay on it in 1858. Hebra explained the brilliant results in 1861, by the permanent maceration of the skin and sloughs, by the prevention of formation and absorption of pus, by the relief of pain and by the prevention of loss of heat caused by the destruction of the epidermis. Hebra's son, in 1877, denied an effect of toxemia upon the symptoms, but Passavant had already expressed his views thus: "The continuous bath presents conditions the effects of which have not yet been made out, but which, however, favor the process of healing. The uniform warmth and pressure of the water are of value. Undoubtedly, metabolism also is increased when compared with that of the healthy skin, as endosmosis is augmented;" and we now know that Passavant was right. Referring for further details to an essay on "The Continuous Bath," in the *Medical Brief*, August, 1905, the cooler temperature of the bath in the very beginning will not only relieve pain, but, as already stated, also powerfully stimulate the system when succumbing to shock. The bath, as continuously used, *i. e.*, somewhat around 90° to 95°, F. is an invaluable regulator of the circulation and, therefore, greatly helps to correct defects in circulation, stagnation and prevent thrombi, and restores the normal tone of the vessel walls. It increases metabolism and must aid in excreting the pro-

ducts of destruction and toxins. Finally, the bath prevents abnormal loss of heat, as it is in our power to keep the water at a temperature at which loss of heat is impossible.

Concerning the wounds, the local effect cannot be too strongly emphasized, as all observers unanimously state, that incident to the maceration of the epidermis and sloughs, infection is made nearly impossible; lymphangitis, erysipelas and pyemia are prevented, absorption of toxic products is ideally avoided, and the sloughs loosen more quickly. Altogether, it is not saying too much to state that, although statistics are not conclusive and are impossible, the number of deaths after burns must be decidedly reduced, especially in burns of the first and second degrees.

Sonnenburg found that animals remained alive after extensive artificial burns, if the spinal cord had before been cut, so that the over-excitation could not be transmitted to the central organs and the blood pressure did not fall. Valentin and Schiff found that varnished animals lived if they were kept warm and loss of heat thus prevented. Both phenomena are provided by the water treatment.

As the third aim in the treatment of burns, I mentioned the prevention of injuries due to irrational manipulation. I do not hesitate thus to qualify the procedure of our surgeons, as outlined, for instance, in Eulenburg's "Real-Enzyklopädie:" "In green and extensive burns a thorough cleansing should be done, if possible, under narcosis, by means of water, brush and soap and followed by disinfection with ether or weak disinfecting solutions." Nothing more clearly illustrates to what indiscriminate bacteria-hunting may lead than the fact, that we can hardly imagine a method better adapted to remove precious layers of epidermis, to reduce the vitality of the remaining cells and to eradicate the remnants of epithelial cells that may be left in the fundus of a gland. Not only this: the whole procedure has often to be done without narcosis and is then an ordeal comparable only to the mediaeval tortures for expelling evil spirits. Next to this professional criticism stands that of laymen, who covered burns with grated potatoes, sauerkraut, clay, crushed fleshy plant leaves, milk, flour and other substances more pretentious than water; all these act by cooling or moisture. To the long line of queer stuffs may be added those which are

taken with or without a doctor's prescription: alcohol, ether, turpentine, and carron oil, which even a surgeon has termed *filthy*. They all have the disadvantage of bringing into contact with delicate, unprotected cells, chemicals which are caustic, or decompose the protoplasm, and, in short, reduce the vitality of the epethelium.

In what follows we shall show that our procedure is strictly in accordance with the laws of physiology, pathology and bacteriology.

In addition to what has been already mentioned, we may state, further, that contact with the cool water of the bath or compress quickly restore the lost tone of vessel walls. Thus, waves of normal blood are forced through the afflicted area and provide materials of reconstruction. We know from pathology that the alterated wall of vessels is quickly restored by normal blood, and the experiments of Winternitz and others show, that the rational application of cold is followed by a decided increase of the red and white blood corpuscles and of hemoglobin. In this way, for instance, under a compress, the vitality of a burn is augmented, and thus it can be understood that blisters up to half an inch in diameter are absorbed during a proper bath or compress within one hour or so, and the appearance of new blisters is positively impossible. The primary layer, as I named it, does not interfere with this result, which is brought about exclusively by thermic factors, acting through the thin covering. This also does not cause retention of inflammatory products, as these are not yet developed and are constantly absorbed by the changed compress. If, later on, the wound should smart, it is advisable to place on it one layer of absorbent gauze for a longer time, and to perform all manipulations over this. Experience shows that pain, when the compresses are changed or an occasional irrigation is given, may successfully be avoided without disturbing the effect.

For further reasoning, I shall quote what I wrote in the *New Yorker Medicinische Monatsschrift*, October, 1902:

" In regard to the principles of the treatment of wounds, it is generally recognized that that procedure is the best which, firstly, prevents infection, secondly, most thoroughly removes secretion; thirdly, reduces irritation as far as possible, for the

secretion will be diminished in proportion to it; and fourthly, the treatment should aid and favor as far as possible the delicate microscopic processes that occur in the wound and lead to recovery."

Powders and ointments do not fulfill these demands. First they dry up, and then form a shield, under which all conditions for germ culture are given. Ointments check the removal of secretions more than they favor it. The disinfectant contained in them does not act as reliably as powder or solutions and both do nothing in regard to the fourth factor mentioned, which is to turn the scale in favor of the water treatment. The undesired by-effects of powders and salves have always induced surgeons to prefer the wet bandage method, which is praised by Tillmanns, Stoneham, Watson-Cheyne, Foote and others:

" However this may be, the wet bandage offers, firstly, ths drug in a more efficient state, and, secondly, it thoroughly absorbs the secretions of the wound. Well wrung-out bandagee serve this end better than any dry material, which is rendered more and more impervious as it becomes saturated with secretion. In wet compresses, on the other hand, there is a good deal of diffusion. No matter how this may occur, we shall see that the secretion is reduced with every successive compress. Furthermore, the water of the compress is of greater chemical adequacy to cell protoplasm than vaseline or a fat. To prevent the water from being robbed of its salts, I never use distilled or boiled water, except when the supply is visibly contaminated. Certainly there could be no objection to physiological salt solutions."

The question is, how is a wet compress to be administered, to comply with the conditions mentioned?

First, let us settle the chemistry of the compress. In considering the chemical composition, we must discriminate between the chemical composition proper and the degree of concentration of the lotions. Most authors recommend, first, carbolic acid and corrosive sublimate, then a number of more or less generally applied drugs, of which each prefers a different one. Carbolic acid and sublimate are likely to cause poisoning and even gangrene. As a rule, the profession regards only the bactericidal power, but here it behooves us not to overlook the third condition, that is, that the wound should be irritated as little as possible. One should always bear in mind that an agent, when killing the microbes, or preventing their develop-

ment, will likewise act upon the growing and proliferating cells of the tissue deprived of its natural covering, which consists of superficial layers of cells impervious to water and, to some extent, even to some chemicals. Therefore, I never use carbolic acid, sublimate or silver nitrate solutions, the last of which, as I have observed, produces a more abundant secretion.

It seems to me that the concentration, which is usually recommended too strong, is a factor of still greater importance. On the one hand, authors warn against changing the bandage too often and even exposing the sore to air; on the other hand, they employ uselessly concentrated lotions, thereby lowering the vitality of the superficial cell layers. (Watson-Cheyne, $HgCl_2$ 1:1000 !).

There is no objection, when beginning the treatment, to apply a somewhat higher concentration, but very soon one may obtain better results by saturating the frequently changed compresses with more dilute solutions. I never met with a condition pointing to increase of the bacteria previously existing in the wound, or betraying new infection; on the contrary, the secretion and the general aspect become so favorable that one believes that it is an aseptic wound. Whatever harm is done by the low concentration, is overbalanced by the fact, that the frequently changed compresses bring new quantities of the disinfectant into direct contact with the infected parts. The chemicals I have used for fifteen years are: chinosol, 1:2000; boric acid, $\frac{1}{3}$ to $\frac{1}{2}$ of the concentrated solution; alum. acetas 0.4:100; protargol, $\frac{1}{4}$ %; and if the granulations are too hyperemic, a filtered infusion of sage leaves, diluted to straw color. That such weak solutions are efficacious, is shown by the cauterizing effect of solution of zinc chloride, 1:600. I use this instead of silver nitrate in abundant granulations. The gauze is wrung out of it and applied as mentioned. In from three to six hours it turns the granulations to a thin slough, without pain. Then one of the disinfectants is used. The slough will adhere thereto and easily come off in this way. If the cauterizing effect is not sufficient, I repeat a zinc chloride compress a few times. I have never used a caustic in fifteen years, although I have had to remove granulations of the thickest kind from neglected wounds that came under my care.

Besides the chemical and its concentration, the temperature of the solution is of importance. It should be below blood temperature in order to cause reaction in the wound, as a Priessnitz does over the intact skin; but it should not be too cold, as it irritates the wound; 75° to 85° F. are tolerated without pain. Here again the patient must guide us. Pain during the change of bandage can be avoided by a higher temperature.

The gauze used should be as hydrophile as possible, and of a shape and size to exactly cover the borders of the wound. The healthy skin covered by the impervious stuff, may be protected against maceration by dusting-powder or a little lanoline. Even this is seldom necessary, however, as the gauze should be wrung out so that no water escapes from it under the impervious covering. If the gauze sticks to the wound and causes pain when removed, it was too dry, or the impervious covering did not close perfectly. In rare cases, in extraordinarily painful sores, two layers of gauze may remain on the wound for a longer time, and over these the compress is changed more frequently, until pain is relieved.

The number of gauze layers depends upon the degree of oozing. In the beginning, eight to ten, later four, will be sufficient, as secretion is reduced to such an amount, that the gauze is often tinged only a fine yellow hue. The gauze may be changed every one to eight hours. The best guide is the pain and the degree of staining of the gauze. In the beginning, or in wounds oozing freely, it may be necessary to change every one to two hours, but it is surprising how quickly the secretion is reduced, so that three changes within twenty-four hours often suffice If everything is carefully performed, there should be no pain, either during change of compress, or later.

The impermeable cover may be oiled-silk or any other suitable material. Rubberdam I never saw or heard of in practice. I use it in all head wounds, for which I cut a piece the shape and size of the wound, attaching thereto strips one inch wide, by means of a needle and cotton thread, fitting around the head. This bandage remains and fits ideally. In the same way I proceed with wounds of the thigh and, frequently, patients have continued working. For instance, a mask was kept around the head by three strips with common hooks and eyes, and a chest wound was covered by rubberdam, to the

borders of which a number of hooks and eyes had been attached, so that the covering could be easily changed by opening and closing the hooks on the back.

If the location of the affected part allows of bathing, two or three partial baths during twenty-four hours may be given. If inflammatory pain is present, 85° to 90° will be the best; if no pain is present, but there is some inflammatory redness of the borders of the wound, the same temperature, or 100° to 105°, is advisable, as in non-inflamed cases. To explain this, I quote the experiments of Schaeffer: He passed through the skin threads soaked in carbolic acid, sublimate of mercury or bacteria cultures and found that heat accelerated the blood stream, so that stagnation in the capillaries and accumulation of blood elements, as pathology teaches, were prevented and healing was quicker. Penzo found, in Bizzozero's laboratory, that after prolonged hot baths, the number of mitoses in the epithelial cells was largely increased. Both facts point to a highly improved physiological progress in the wound. I quote what I wrote in the *N. Y. Med. Monatsshrift.*

" Heretofore, the surgeon has feared and hesitated to change the wet dressing, but he has been finally forced to do so by the accumulation of secretions."

Even the frequent change, if done under strict observation of the given rules derived from hydrotherapy, is a valuable factor, as it favors circulation in the wound and causes the physiological processes leading to recovery to progress as speedily as nature allows. What Watson-Cheyne says (to cite an author incidentally at hand), is not true:

" Sores left by burns heal much more slowly than open wounds made by the knife, probably because the heat not only destroys the vitality of the part immediately acted upon, but also impairs that of the tissues around, so that, in the early stages, the vital processes in them are not nearly so active as usual."

It is in our hands to positively change the condition altogether.

To illustrate the result of the hydriatric management of burns, I refer for further details to the case (Fig. 1), which I presented to the German Medical Society and published in the *N. Y. Med. Monatsschrift* for October, 1902.

Fig. 1.—Burn of breast, face and hands of the third degree, on July 7, 1902. Photo taken 1908 (Grosse)

The boy, four years of age, set his clothes afire on July 7, and burned his face, neck, chest and both hands, the stratum papillare being entirely destroyed. The face, chin and both cheeks were burned up to the temples, except the median parts; the borders of the ears were gone and so was the region behind the ears, and the upper parts of the front of the neck down to the bending fold. No epithelium was left, so that epidermization had to proceed entirely from the border. The same condition existed with the chest wound.

When the case came under my treatment on August 6, the wounds were covered with granulations 0.3 to 0.5 cm. high and profusely oozing. The chest wound was closed in six weeks and the face wounds in ten weeks, without skin-grafting, which had been regarded as absolutely necessary by my predecessor. The treatment was done at home without a trained nurse.

Foote writes in his textbook on Minor Surgery:

"An epithelial edge will grow about an eighth of an inch a week. A granulating area, therefore, which is an inch in its smallest diameter, will require a month for its complete repair. Areas larger than this should be skin-grafted."

The illustrated case shows that we may cover even larger areas of wounds more quickly by this method, without skin-grafting, which we will seldom have to resort to. It must also be mentioned that the scars are softer than in other methods, as the granulations are decidedly less, in fact, so slight, that we seldom need resort to caustics.

To conclude: The hydriatric management of burns is partly an alteration of the long-known wet-bandage method, according to hydrotherapeutic principles. It is superior to all other methods in stilling pain, saving lives and securing good results; it is easily performed, cheap and can be undertaken everywhere. I heartily join in the hope of Dzondi and Winternitz, that hydriatric management of burns will be the treatment of the future for the sake of suffering humanity.

SUBINVOLUTION.

BY SAMUEL WYLLIS BANDLER, M.D.
Adjunct Professor of Diseases of Women, New York Post-Graduate Medical School and Hospital.

In pregnancy the uterus undergoes great hypertrophy, hyperplasia and dilatation, associated with which is a stretching, a growth and hyperemia of the broad and other ligaments, and of the various pelvic structures. In addition, there is hyperemia and congestion of the intra-abdominal organs. The intra-abdominal organs and the abdominal wall are subjected to pressure and stretching by the enlarged uterus. The vascular system is put to greater strain and a general strain of hyperemia and altered metabolism results. During pregnancy we have an increased amount of watery elements of the blood, an increased proportion of fibrin, a diminished amount of albumin, an increase in the white blood cells, a relative diminution in the number of red blood cells and in the amount of hemoglobin. Before labor the temperature is higher in the last three months of pregnancy, and there is an increase in the elements of the body, equal to one-thirteenth of the body weight. This increase is due to serous infiltration, and to the increased ability of the body to form organized tissue. Post partum, after a temporarily short rise, the temperature is lower, the blood pressure sinks and becomes normal on the sixth day. After labor there is a diminution of tissue-change and a diminution in the amount of urine.

The return to normal size, position and condition (1) of the pelvic structures, (2) of the abdominal organs and of the abdominal wall, (3) of the general circulatory apparatus and of the nervous system, constitutes involution. This is a process which, for its completion, involves from three to six months and which often never occurs. The failure of involution (subinvolution) may be due to frequent successive pregnancies. It may result after one labor.

Uterine Subinvolution.—During uterine involution, fatty degeneration and atrophy of the muscle-fibres occur so that the muscle-fibre may regain its normal dimensions. Failure of uterine involution is often due to congestion in the uterus or

in the surrounding tissues. Displacement of the uterus is an important factor. Retained placental structures or retained decidua is another contributory element. An important element is a poor general circulation. Uterine subinvolution is most frequent in women who do not nurse. Within a few weeks after labor the uterus is large, soft and atonic. The patient has a sensation of weight in the pelvis; there may be profuse serous discharge, there may be irregular bleeding. There is backache and atony of all the pelvic structures. If such a condition is allowed to persist, permanent changes take place.

Subinvolution Fibrosis.—Under the peritoneal covering of the uterus is a membrane of elastic fibres. In addition, between this layer and the mucosa there are three other layers of elastic fibres. The elastic and fibrous tissues situated between the muscle bundles send branches between and around every muscle cell at right angles to the long axis of the cell, furnishing a frame work or elastic support which protects the fibres from overstretching, and which permits their return to normal position on contraction. In pregnancy, the elastic fibres undergo hyperplasia. The same change takes place in the parauterine and peri-uterine elastic fibres. When involution fails to occur and subinvolution persists for a long time then, in addition to subinvolution of the muscle fibres, there occurs a marked increase in the elastic and fibrous connective tissue. There finally results an end-stage, which furnishes an enlarged, hard, brittle uterus associated with which is an inelasticity of the blood vessels. In such a *fibrosis* there is hyperplasia of the connective tissue, the elastic elements are thickened, and there is an increased amount of fibrous connective tissue. In the connective tissue around the uterus, in the broad ligaments, in the connective tissue between the bladder and uterus, there are large dilated veins. In the broad ligaments there are plexuses of varicose veins. The tendency to the occurrence of fibrosis is increased by numerous labors occurring in rapid succession; by retroflexion occurring after labor; by inability to nurse, and by the performance of physical labor. As a result of the subinvolution and of the frequent pregnancies, such uteri are hard, enlarged, firm. They grate on cutting. Such uteri have poorly conditioned muscle fibres and are composed of

much fibrous connective tissue and of numerous elastic fibres, poor in contractile power. This uterine alteration of itself, however, is purely a local phenomenon, and causes very frequently toward the age of the normal climacterium a lack of contractile power in the uterus, with or without arteriosclerosis of the vessels, which results in profuse bleedings, for which, often enough, only a hysterectomy can be of any aid.

Inflammatory Metritis.—In contra-distinction to such a simple subinvolution fibrosis, the uterus may be thickened, hypertrophied, sclerotic or enlarged as a result of chronic inflammatory changes in the uterine lining and uterine wall.

We are then dealing with an inflammatory end-stage of a real inflammatory metritis. As a rule, the distinction is not made between such alterations due to inflammation and similar changes due to failure of involution on the part of a uterus. The latter is generally considered under the heading of metritis. If the term metritis is to be used for inflammatory cases, then the end-stage of subinvolution should be called non-inflammatory metritis or fibrosis uteri.

Pelvic Subinvolution.—Subinvolution of the uterus itself is often only of secondary importance. Of far greater significance is the condition, size and elasticity of the ligaments about the uterus. These ligaments filled with elastic connective tissue and muscle fibres are responsible in a very great measure for retaining the uterus in its normal position. The elastic fibres and the connective tissue about the vagina, bladder, etc., are of great importance in retaining these two organs in their normal situation. There occurs very frequently after one or more labors failure on the part of these ligaments to return to their normal elastic condition. They remain elongated, inelastic, flabby, resulting in descent of the uterus, retroversion and retroflexion, descent of the posterior wall of the vagina, descent of the vagina, etc. Of great importance in this connection is the condition of the levator ani muscles and of the muscles in the perineum. Much has been said of the effect of perineal laceration in the production of cystocele and descent of the uterus. It is a fact that even a deep median laceration of the perineum which does not tear the insertion of the levator ani fibres, is not productive *per se* of descent of the vagina. That with lateral laceration such conditions do occur, can be readily understood, when we

consider the insertion of the levator ani fibres to the ramus of the pubis, their close anatomical relation to the lateral walls of the lower part of the vagina and their insertion about the rectum, anus and coccyx. Subinvolution or laceration of these muscles, which form so important a part of the pelvic floor, loosens from their situation the bladder and vagina. Alterations of position on the part of the bladder, uterus and vagina are as much due to inelasticity and atrophy of the connective tissue as to injuries to the muscle fibres.

Ptosis.—The condition which we finally observe constitutes a ptosis of the genital structures, to which the name of hysteroptosis may well be given. The name hysteroptosis is of special significance for, in the majority of these cases, there is also a ptosis of other abdominal structnres. It is natural to expect that an abdominal wall, which does not return to a normal condition of elasticity, in which the recti are abnormally overstretched, in which the fasciæ of the abdominal wall are overstretched, will fail to give proper intra-abdominal pressure, and in this way fail to aid in substaining the intra-abdominal organs in their normal position. This is not however, the only important factor. The same failure on the part of the ligaments of the intestines and stomach to return to their normal state of elasticity; the inelasticity, or atrophy of the connective tissues; fibrosis of the elastic fibres in these structures, and the disappearance of fat about the kidneys are important elements which cause enteroptosis, gastroptosis and nephroptosis, constituting together a splanchnoptosis. Probably half of the cases of enteroptosis in woman have retroverted retroflexed or descending uteri. With or without relaxation of the abdominal wall, with or without displacements of the intra-abdominal organs, there exists, too, for weeks, months and often for years, a condition of intra-abdominal congestion, in which the important blood channels of the abdominal cavity are loaded with stagnated blood, with failure of proper blood supply of these important secretory organs and so resulting in altered functions of the stomach and intestine in particular. Associated therewith, is a heavy dragging sensation, indefinite pains in the stomach and abdominal cavity, feeling of weakness, indigestion, constipation and malnutrition.

Constitutional Subinvolution.—This condition, while a local

one, is markedly dependent upon a lack of tone in the general circulatory apparatus. This is a state which occurs not only in women who have been pregnant but is also a general condition, often referable to rickets in childhood, to the effect of various diseases, to the influence of chlorosis and anemia. A condition of this sort is very frequently acquired by women who have been pregnant, and it is, of course, especially aggravated in those patients in whom the etiological factors just mentioned have been present. There is improper circulation of blood throughout the entire system; there is a failure of proper nutrition of the various organs of the body; there is improper oxidation of tissue; there is a sensation of general languor and lassitude, a lack of energy and tone. Combined with this general circulatory lack of tone, is a parallel condition in the nervous system. There is physical and mental asthenia; in other words, a neurasthenic state is the predominant condition. These patients suffer from palpitation of the heart, are weary and tired; suffer from indigestion and constipation; sleep is disturbed or irregular, and there is restlessness. Patients cry easily and show a lack of mental poise and stability. This condition may be well described as one of constitutional subinvolution.

According to Abrams, "There are a large number of gastric and intestinal affections with bizarre and protean symptoms, designated as gastric and intestinal neuroses, but which in reality owe their genesis to congestion of the intra-abdominal veins. The greater the intra-abdominal tension, the less blood will be contained in the abdominal veins. This tension is largely dependent on the tone or tension of the abdominal muscles. Therefore, nervous exhaustion is a frequent cause of diminished tone of the abdominal muscles, which in turn diminishes intra-abdominal tension and conduces to blood stagnation in the veins of the abdomen. Venous congestion interferes with a proper supply of arterial blood. The tissues and the organs bathed in pools of stagnant blood are practically in a state of asphyxia. The toxic products of digestion, which are normally removed by an unimpeded circulation, have a specifically poisonous effect upon the sympathetic system; a fact which is evident, owing to the frequent occurrence of depression, prostration, and nervous symptoms in nearly all disorders of the ali-

mentary canal. Gastro-intestinal disturbances of whatever nature, seriously compromise the integrity of the nervous system, either by inducing neurasthenia or aggravating it if it exists."

Splanchnic Neurasthenia.—The entire question of splanchnic neurasthenia is one of abdominal plethora dependent on a variety of causes, notable diminished intra-abdominal tension, insufficient lung development, a defective vascular apparatus. Splanchnic neurasthenia is one of the few forms of neurasthenia amenable to permanent cure, by measures having for their object relief of abdominal venous congestion. In any splanchnic neurasthenia existing as an independent affection, the relief of symptoms almost positively follows relief of the venous abdominal congestion.

There is certainly a predisposition to the occurrence of abdominal or constitutional subinvolution. It is noted in women in whom there is a general inelasticity, an almost complete absence of fat and a tendency to neurasthenia. Such women frequently have little tone. Even the greatest subsequent care in the post partum stage fails to restore the various organs of the body to a normal state. This general state often occurs without the presence of an enlarged uterus. In fact, prolonged nursing is a frequent cause.

Lactation Atrophy.—The changes occuring in lactation-atrophy are an eccentric atrophy of the uterus with a cavity of normal size, but with a deficiency of muscular elements. These latter cases are frequently associated with small adnexa. The majority of nursing women who have a uterus under the normal size, show all the evidences of poor nutrition, and especially laxity and flabbiness of the general body structures. In "prematurely aged women," lactation is poorly borne. It is in these cases that Fromell finds the greatest amount of uterine atrophy, and he supposes it to be an evidence that nursing deprives the body of a large amount of nutrition.

Thorne considers lactation-atrophy to be a reflex trophoneurosis, and believes that every nursing amenorrhoic woman has a hyperinvoluted uterus, without, however, an involvement of the ovaries. He acknowledges the frequency of anemic conditions associated therewith, but observes that those cases menstruating during nursing show no atrophy of the uterus. This associated menstruation is an evidence of sufficient ovarian stimulation.

These conditions of abdominal and constitutional subinvolution explain, in many cases, the relation so long, and even still, held to exist through reflex channels between cervical lacerations, cervical erosions, cervical catarrh, uterine catarrh, and especially uterine retroversions and flexions on the other hand, and certain nervous phenomena, on the other. In many instances the nervous condition is due to alterations in the ovary produced by tubo-ovarian, ovarian and peritoneal complications, which complications are generally the result of an upward extension of a cervical infection. These ovarian lesions may be simply that mild infection of the follicles which leads to the formation of socalled "cystic ovaries." The ovarian changes may be sclerotic in their character, resulting from mild tubal and peritoneal infecting lesions. The inflammatory changes in the ovary may be chronic in form, producing alterations not always easily classified but interfering with the secretory work of these glands. Aside from inflammation, congestion and improper circulation may interfere with the proper function of the ovaries. Hence, pelvic and abdominal subinvolution may cause such functional ovarian changes as are symptomless, so far as pain or marked pain is concerned. Even changes in the menstrual regularity or in the menstrual amount, may be slight or absent, through changes from normal are often present. The important end-effect is interference with the proper secretory function of the ovaries.

Hyperthyroidism.—Hence we are often concerned with the care of patients who have grown nervous since child-birth. Of course, among such instances are nervous conditions due to various other causes and tendencies. A certain proportion of such nervous patients undoubtedly suffer from what is known as hyperthyroidism. Cases of hyperthyroidism may occur without evident local thyroid changes upon which the tangible responsibility can be placed. These are due to faulty relation between the ovarian secretion, on the one hand, and the thyroid secretion on the other. Altered or diminished ovarian secretion results in a relative increase of secretion from the thyroid. The consequent symptoms vary from nervousness and irritability, up to symptoms which include sleeplessness, palpitation, rapid pulse, causeless diarrhea, flushes, etc., but without any exophthalmos, goitre or marked tachycardia; or

else exophthalmos may be of varying degrees, or goitre may be more or less marked. It is important to recognize the fact that Basedow's disease frequently occurs in an aberrant form, and that the cases shade gradually into forms which, since exophthalmos, goitre and marked tachycardia are absent, are to be called hyperthyroidism. Many such patients are found to have cystic ovaries, sclerotic ovaries or other pathological non-recognizable ovarian changes. Thus, relative hyperthyroidism produces a group of symptoms like the nervous symptoms of the typical menopause or climacterium, except for the fact that flushes or flashes are less marked or absent.

Post Partum Treatment and Prophylaxis.—A weighty element in the causation of these conditions of subinvolution and hyperthyroidism is failure of proper care in the post partum stage. In the post partum stage frequent examination of the patient should be made, and the uterus and its ligaments should be restored to the normal. Abdominal massage should take care of the abdominal walls; deep abdominal massage should take care of the stomach and intestines. Tonics should take care of the blood corpuscles and stimulate the circulatory apparatus, and baths should restore the circulation of the nervous system to its proper balance.

Within a few hours after labor the patient should be encouraged to spend very little time on her back, lying mostly on the right or left side. After the flow of milk has been established and the uterus is in the true pelvis, the patient should spend an hour every morning and an hour every afternoon lying flat on the stomach and should sleep in this position a part of each night. A tight abdominal binder is by no means advisable; a comfortable bandage being all that is necessary. Attention should be paid to avoiding distention of the intestines by gas. At all times the bladder should be emptied at intervals of six hours. From the fifth day on patients should have a commode at the bedside. Patients should be allowed to sit up on the fifth or sixth day for a short time, increasing the period daily so that the patient can be out of bed on the eighth to the tenth day. Toward the end of the second week or at the beginning of the third week, a course of baths should be begun, containing at first salt, then salt and calcium chloride and, finally, carbonic acid gas should be added. Internal examination should then

determine the position of the uterus. If there is any appreciable retroversion or retroflexion or a noticeable tendency to descent of the uterus, a pessary should be introduced; or else the vagina should be packed three times a week with gauze and glycerine, to give the uterus and the connective tissue elevating support. The return of the uterus to its normal size should be aided by the administration of ergot after the flow of milk is once well established. Lifting should be avoided. If the abdominal walls fail promptly to return to a tonic state, superficial and deep abdominal massage should be given and an abdominal belt of silk should be applied and worn. Various exercises should be taken. The sinusoidal electric current should be used. Constipation should be corrected.

Treatment should be devoted rather to the prevention of these conditions than to their cure, for if once the uterus undergoes a fibroid change, if the ligaments of the pelvis and of the abdomen are not early restored to normal tone, treatment is then concerned with giving support by mechanical apparatus to the uterus, to the abdominal walls and to the intra-abdominal organs. When care of the general circulation is begun early and done systematically shortly post partum, its application is extremely efficacious in avoiding either constitutional subinvolution or hyperthyroidism. The longer this condition exists before treatment, the more difficult it is to correct; for the patients lose their courage, acquire ideas and phobias, and, since the resulting condition came on after labor, are firmly convinced that a genital condition is at fault, and feel certain that operation only will be of any value. It is just in this class of cases, where operations done for the correction of displacements of the uterus, cystocele and perineal lacerations, for fixation of movable kidneys, etc., fail of their proper purpose, because such operations do not cure the basic general asthenic condition of the patient.

In the treatment of these cases coming to us with pelvic, abdominal and constitutional subinvolution already established, attention must be paid to the size and position of the uterus, the character of the uterine ligaments, the abdominal wall and the organs within the abdomen, the general circulation, and the condition of the nervous system. So far as the uterus is concerned, we must make use of the glycerine treatment

with support either by gauze packed into the fornices or by the pessary, supplemented by the use of hot vaginal douches. As regards the abdominal walls and ptosis of the intra-abdominal organs, we make use of abdominal massage with diet for the correction of obstipation; of exercise directed to the strengthening of the abdominal walls; of the abdominal belt or Rose bandage, and of hydrotherapy, most easily administered in the form of the Nauheim baths. The sinusoidal current is valuable. So far as the use of drugs is concerned, those given for the effect on the nervous system or to influence the hemoglobin of the blood are indictated. For the former purpose, a combination of bromide of strontium and sodium glycero-phosphate is the best. For the latter purpose, a combination of iron, arsenic and ovarin is of the greatest value, often combined with cascara.

These cases are truly benefited by the administration of ovarin, iron and arsenic and by conservative local treatment. A most important aid is furnished by the administration of the Nauheim baths, care being taken to avoid, in extreme cases, even a temporary excitability of the nervous system. Restoration of a normal circulation is followed by benefit, and most assuredly do the ovaries share in this nutritional alteration.

PYURIA IN WOMEN.

BY HENRY DAWSON FURNISS, M.D.
Instructor in Diseases of Women, New York Post-Graduate Medical School and Hospital.

Pyuria in women and men has many points in common, but the anatomy of the two genito-urinary systems is sufficiently different to furnish some etiologic factors that are present in only one of the sexes. Urologists devoting their studies mostly to diseases of the genito-urinary organs in men, claim that inflammatory troubles of the urinary tract, especially cystitis, is more common in women, while gynecologists are of the contrary opinion. It is claimed that the short urethra of the woman and the close relation of the bladder to the uterus, tubes and ovaries render it more liable to inflammation than the bladder of the male. On the other hand, the very shortness of the female urethra, so seldom the seat of obstruction, gives better drainage than the male urethra, whose lumen is so frequently narrowed by an enlarged prostate or stricture.

In speaking of pyuria, not all cases of purulent urine should be included; for vulvo-vaginitis, vulvo-vaginal abscesses, endocervicitis, endometritis, and abscesses discharging into the vagina may all furnish pus to the urine. Only those cases in which the pus is from the urinary tract are considered. This would include those in which the pus was from the urethra, with its Skene's ducts, Littre's follicle, and crypts; the bladder, the ureters, the pelves of the kidneys and the kidneys; abscesses discharging through any of these are properly included.

Until the introduction of the endoscope and cystoscope, diagnosis of the seat of the various lesions of the urinary organs depended upon the clinical history, the physical examination of the patient and a careful study of the urine. Then, and now, these often furnished a comprehensive and accurate means of diagnosis. In tubercular cases instrumentation is usually contra-indicated and not to be employed unless the knowledge to be derived is essential and not to be gained by other methods. In young women suffering from frequent and painful micturition with or without pyuria, the idea of tuberculosis immediately comes to our mind, especially when we can eliminate gonorrhea

and instrumentation as etiological factors. In these cases a most careful examination should be made for the tubercle bacillus, and this failing, innoculation of the guinea-pig should be tried.

Some authors do not include in the consideration of pyuria the condition known as bacteriuria, but as a greater or less amount of pus is generally present, and as the condition is due in the majority of the cases to a mild pyelitis, it seems best that it should be considered in the subject of pyuria. While the colon bacillus is the microörganism usually present, the typhoid bacillus and the staphylococcus have been observed.

Whenever pus is found in the urine, our first effort should be to locate its origin and determine the etiological factor; the majority of the inflammatory disorders of the genito-urinary system recover spontaneously after removal of the cause.

When the pus is from the urethra it can usually be seen escaping from the external meatus, or by pressure can be expressed from the canal. The most frequent cause of purulent urethritis is gonorrhea; this can be readily determined by a microscopic examination of the pus. After the acute attack has subsided, pus is at times discharged from Skene's glands, Littre's follicles and the crypts of the canal. There is a condition known as sub-urethral abscess, or abscess of the urethro-vaginal septum, which produces an enlargement beneath the urethra and which intermittently discharges pus into the urethra through a narrow opening on its floor. Howard Kelly studied one of these microscopically and came to the conclusion that it was a dilated follicle.

The bladder is the common reservoir for the urine from both kidneys, and in cases of pyuria it is our endeavor to determine if the pus is produced in the bladder, or is deposited here from other sources. To this end we are aided by the history, past and present; the examination of the patient; the study of the urine; bladder-washing; cystoscopic examination and ureteral catheterization.

In the acute cases, as a rule, there is not much difficulty in locating the source of the pus, for then local symptoms are sufficiently intense to point to the origin of the trouble. In urethritis there is the discharge and the very painful urination; in cystitis the painful and frequent urination, with a sense

of heat and fullness, and tenderness over the bladder; while in kidney cases there is pain in the region of the kidneys, possibly tumor and at times more or less temperature. In cystitis, painful and frequent urination is more marked than when the pus is from other sources; the bladder can be the reservoir for pus from other regions without itself becoming infected. In tuberculosis of the kidneys, the urine is particularly irritating, and gives rise to bladder disturbance without the actual production of cystitis. The history of instrumentation or of operation in the immediate neighborhood of the bladder shortly preceding any urinary disturbance, would point to the bladder as the organ giving rise to the symptoms.

If there is pus in the bladder urine; if the bladder is catheterized and thoroughly irrigated, and a second specimen obtained by means of a catheter an hour later contains the same amount of pus as the first, the inference is clear that the pus is derived from a source outside the bladder.

In the examination of the urine it will generally be found that, after standing, the deposit of pus in cystitis cases is not so compact as when the pus is from the kidney, and that the deposit is covered by a more or less flocculent mass of mucus. In inflammatory diseases of the kidney, the urine is of lighter color, with a larger albumin-ratio in proportion to the amount of pus than in cystitis, and the pus is deposited in a solid mass, like sand, with the supernatant urine cloudy.

With the aid of the cystoscope, the nature, the extent, and often the causative factor of the lesion to be dealt with can be determined. As before mentioned, instrumental examination is not to be used in tubercular cases except to determine the necessity or advisability of operation; it is justifiable when nephrectomy is contemplated and it is desirable to determine if one or both kidneys are involved, and if only one, the functional capacity of the other. In cystitis it is often observed that the inflamatory process is not general, but localized, and when stones have to be dealt with, there is no better method of examination than cystoscopy when applicable. In very large stones and small bladder capacity, the sound is the better instrument. In the contracted bladders met with after long standing cystitis, or vesical fistulas of long duration, the use of

the cystoscope is unsatisfactory, and at times impossible. Where a neighboring abscess is discharging into the bladder, the point of communication can be determined through a cystoscopic examination.

When the inflammatory process is above the bladder, and the source of the pus has not been located by other methods of examination, cystoscopy and ureteral catheterization furnish most valuable aid. Frequently the appearance of the ureteral discharge, the ureteral mouth and the surrounding bladder mucous membrane are sufficiently characteristic to enable one to diagnosticate the origin of the pus, and at times the type of lesion present without ureteral catheterization. When pus is descending from the kidney in large amounts, it can be seen escaping from the ureter either as a very turbid spurt, or, when there is little urine from the kidney of that side, as a small rope of pus that reminds one of sausage passing from the grinding machine. The urine from the ureter can often be collected direct through the Kelly speculum that has its distal end cut away obliquely. In pyelitis of any severity, there is pouting of the ureteral meatus, and enlargement of its lumen at times. When there is injection of the bladder mucous membrane around the mouth of the ureter, and especially when this is combined with a patulous, displaced, and ulcerated meatus, a destructive lesion of the kidney can be diagnosticated; the added presence of tubercle bacilli in the urine completes the diagnosis of destructive tuberculosis of the kidney (Fenwick). When the amount of pus in the urine is small, it is often necessary to resort to ureteral catheterization to determine from which kidney it arises.

The inflammatory diseases of the kidney, or those accompanied by pus, are catarrhal pyelitis, suppurative pyelitis, pyelo-nephritis, pyo-nephrosis, and abscess of the kidney. In all these the thought of stone or tuberculosis must be constantly present, and with these eliminated, search should be made for evidence of back pressure in the kidney, either from obstruction to the urethra or to the ureter. While we do have cases of hematogenous infection of the kidney, a large majority follow retention of urine in the kidney pelvis. Among the causes of ureteral constriction may be mentioned the kink frequently occurring in movable kidney; pressure of the fibroid or pregnant uterus; ovarian cystomata; prolapse of the uterus; involvement of the

ureter in the scar of a vesico-vaginal fistula, and quite often involvement of the ureter in cervical and uterine carcinomata. A large percentage of the direct causes of death in carcinoma of the cervix and uterus is due to uremia or kidney infection from ureteral obstruction.

In simple pyelitis the amount of pus is small, and there are no casts to indicate the involvement of the parenchyma of the kidney; in the suppurative form the amount of pus is larger, and there is always a large amount of albumin, and frequently numerous casts of the hyalin, granular, epithelial and pus type to indicate extension to the kidney substance. In pyo-nephrosis the amount of pus is even greater, the amount of urine smaller, and the discharge of the pus is intermittent; during the time in which the pus is retained the constitutional symptoms are more marked, to be relieved by the escape of the pus.

In abscess of the kidney there are intermittent discharges of pus, pain, tenderness and tumor mass in the kidney region if the abscess is large, and constitutional symptoms of confined pus.

Dr. Edwin Beer, of New York, found that methylene-blue, given by the mouth, was found in some of the pus cells of the urine as late as two and a half years after administration in a patient who had small abscesses in the kidney. He is of the opinion that the methylene-blue had entered into, and remained in combination with the nuclei of the pus cells and that this pus was discharged when the small focus in which it was contained had ruptured. With this experience in mind, he was able, by the delayed excretion of blue-colored pus cells, to diagnosticate abscess of the kidney in a case at first thought to be simple pyelitis. Both diagnoses were confirmed by operation. The objections to the method as one of diagnostic aid are that it is not uniformly constant; that the blue-colored pus may not be discharged for an indefinite time, and that it may be discharged at a time when the attendant is not on the lookout for it.

REPORT OF A CASE OF CHRONIC SPLENOMEGALIC POLYCYTHEMIA.

BY ARTHUR F. CHACE, M.D.

Adjunct Professor in Medicine, New York Post-Graduate Medical School and Hospital.

The attention of the medical profession was first called to the condition known as chronic cyanosis with polycythemia, by H. Vaquez,[1] in 1892. Since that time the condition has sometimes been spoken of as Vaquez's Disease. Eleven years later Osler[2] reported nine cases of this affection, of which five cases were collected from the literature and four were personal observations. The following year the same observer summarized 17 cases[3]. In the past five years about forty cases have been reported.

Although several articles have recently been written upon this interesting disease, there is so little known concerning the condition that it was thought advisable to publish the following case, together with references to the recent literature on the subject.

Miss E. W., age 17 years; schoolgirl; born in New York State; was referred to the New York Post-Graduate Hospital by Dr. Cora M. Ballard. The patient was admitted to the medical wards, August 19, 1907, and was under observation until September 24, 1907, when she was discharged. Her family history was negative. The father, mother, four brothers and two sisters were living and well. Aside from measles in childhood, she had always been in good health. She takes a glass of beer or whisky occasionally, and also drinks two cups of coffee for breakfast, and averages two cups of tea each week. Menstruation has never been established.

History of Present Illness.—Three years ago she was confined to

1. H. Vaquez: "*Sur une forme speciale de cyanose s'accompagnant d'hyperglobulie excessive et persistante,*" *Compt. rend. d. la Soc. d. Biol.*, Paris, May 7, 1892.

2. William Osler: "Chronic Cyanosis with Polycythemia and Enlarged Spleen. A New Clinical Entity," *Am. Jour. Med. Sc.*, Aug., 1903.

3. William Osler: Polycythemia, *Brit. Med. Jour.*, Jan. 16, 1904.

the house for about one month with a severe cough. Since this attack she has suffered from palpitation of the heart, hoarseness, general weakness, blueness of the lips and fingers, and an irritable disposition. Three months before entering the hospital she had a fainting spell, preceded by a distinct aura, consisting of groaning and twitching of the muscles of the forehead. Consciousness was lost for about five minutes, and on awaking the bed clothes were saturated with blood, which had flowed from the nose. During the following three months she had four similar attacks, all of which were accompanied by an aura and severe epistaxis. On admission to the hospital the patient complained, in addition to the above mentioned symptoms, of headache and a feeling of distress in the epigastrium.

Physical Examination.—Patient well proportioned and fairly well nourished. The skin was very dusky and slightly pigmented. Closer inspection revealed dilated veins and venules. The lips, gums, tongue and conjunctivæ were blue. The lingual papillæ stood out prominently. The teeth were decayed. The fingers were slightly clubbed, and the nails incurvated. No superficial glandular enlargements. Heart beat was forcible and accelerated. No adventitious sounds were heard. Marked pulsation of the carotids was visible. Lungs were normal. Examination of the nervous system was negative. Spleen was palpable one and a half inches below the free border of the ribs. Vaginal examination showed an atrophic uterus.

Examination of the blood was as follows:

	Aug. 22	Aug. 30	Sept. 19
Erythrocytes...	7,500,000	7,568,000	5,917,500
Leucocytes.....	6250	4000	5500
Hemoglobin ...	112%	110%	111%
Differential count:			
Polynuclears...	74%	64%	76%
Lymphocytes ..	25%	31%	24%
Eosinophiles....	1%	1%	
Transitionals...		4%	

Systolic blood pressure was 120 mm. of mercury.

Aside from a very slight amount of albumin and an occasional trace of indican, the urine was found to be normal. The temperature and respiration remained about normal. The pulse averaged 100, ranging from 88 to 104.

Course of the disease follows: The patient did not improve

while in the hospital. The most severe symptoms were headache and indefinite abdominal pains. She occasionally complained of palpitation and feeling " out of sorts." While in the medical ward she had one slight convulsion with rather severe epistaxis. Three months after leaving the hospital she wrote saying that she had become quite weak and averaged about two fainting spells a week. The attacks were preceded by an aura in which the eyelids and the skin over the forehead twitched. Usually, consciousness was lost. Sometimes, however, she was aware of her surroundings, but was unable to speak. The attacks were followed by severe headaches. She suffered from attacks of numbness in the left leg. She said that she was growing progressively weaker and more cyanosed.

Treatment.—Potassium iodide was given, in ten grain doses, without any good effects. Oxygen inhalations did not clear up the cyanosis. The nervousness was controlled with mixed bromides. High colonic irrigations of normal saline solution were given daily for ten days, with very little if any benefit. She felt better after the withdrawal of seven and one half ounces of venous blood, but this procedure was discontinued because of her weakened condition. A nonproteid diet was given with the view of limiting the formation of new blood cells.

The disease does not seem to be influenced by drugs. Arsenic, quinine, extract of thyroid gland, mercury, digitalis, adrenalin, erythrol tetranitrate, salicylates, iron, valerian and nitroglycerine have been tried by competent observers without benefit. Exposure of the splenic area to the X-ray has no influence.

The noteworthy features of this case are the age of the patient, the nonappearance of the menses and the epileptiform convulsions with epistaxis. The disease occurs most frequently between the ages of forty and fifty. This patient, therefore, seems to be the youngest recorded case.

Nothing positive is known concerning the etiology and treatment of this affection. Necropsies have been performed upon about fifteen cases. The findings consisted of pronounced engorgement of the organs and intense hyperplasia of the bone marrow. Microscopical examination of the bone marrow showed a predominance of red cells over the normal fat cells. The primary seat of the condition is probably in the marrow of the long bones. The enlargement of the spleen is due to passive congestion.

Osler and Hirschfeld have pointed out that the name Erythemia is more in harmony with the present nomenclature than polycythemia. As leukemia is an hyperplasia of the leukoblastic apparatus, so erythemia is an increase in the growth of the erythroblastic mechanism. Polycythemia is only a symptom, which can logically be spoken of as an erythrocytosis.

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CONGENITAL STENOSIS OF THE PULMONARY ARTERY. A STUDY OF FIVE CASES.

BY ROBERT ABRAHAMS, M.D.
Adjunct Professor of Medicine, New York Post-Graduate Medical School and Hospital.

The study and observation of five cases of congenital stenosis of the pulmonary artery included in this report were carried on at the New York Post-Graduate Medical School and Hospital, in the Medical Clinic of Professor Charles F. Wainwright. They all came under notice within the last five years.

Their points of interest are that three of the patients have reached adult life; one almost passed the period of childhood, and with the exception of the oldest, they all live and do well. Additional items of interest are: first, that in two, there is a family history of congenital malformation of the heart, bespeaking hereditary influence; second; in three, the mothers are rheumatic, suggesting intrauterine rheumatic infection, and, third, that four out of the five patients are males. The diagnosis of pulmonary stenosis was made in each case with due care and reference to the history and physical signs. If other forms of malformation of the heart existed and which only an autopsy could reveal, the predominating signs, however, were those of stenosis.

The following were the deciding symptoms in favor of pulmonic obstruction in all the five cases: (1) a loud blowing systolic murmur having the point of maximum intensity over the pulmonic area: (2) a systolic thrill distinctly felt over the same location, and (3) hypertrophy of the right heart.

That the lesions were congenital, was evidenced from the fact that cyanosis began in early infancy; the terminal phalanges of fingers and toes were club-shaped; dyspnea at rest and more so on exertion and, lastly, no evidence of a lesion in any organ of the body to explain these clinical phenomena.

Case 1.—M. A., male, 25 years old; weight, 155; height, 5 ft. 6 in. Family history negative to tuberculosis, cancer or syphilis. Mother suffered several attacks of rheumatism involving the knees, wrists and ankles. One attack occurred during pregnancy. An uncle on the paternal side died of hemophilia.

Personal History.—The patient was born in a natural way, and nothing unusual was noticed about him until after circumcision—on the eighth day. The same day bluish discoloration of his skin and mucous membranes became apparent. He safely passed through measles, scarlet fever, diphtheria "and a few colds." When he appeared for treatment, he was found to be well nourished, able-bodied and engaged in daily labor—that of umbrella-making. The whole cutaneous surface was distinctly livid; all the visible mucous membranes and conjunctivæ were of a deep purplish blue; the eye-balls bulged; the superficial veins of the chest were preternaturally blue and dilated; the fingers and toes were blue and club-shaped; marked dyspnea.

Examination of the organs of the chest disclosed normal lungs. Considerable enlargement of the right auricle and ventricle. Very little displacement of the apex. The apex beat was strong, aortic second accentuated, pulmonic sound much modified by a murmur. The murmur was loud, blowing and systolic in time; its point of maximum intensity directly over the pulmonic area, with diminishing intensity towards the left clavicle and precordial area. A well-defined systolic thrill was appreciable.

The kidneys, liver and spleen were normal.

Examination of the blood disclosed 8.930.000 red cells, hemoglobin 160%.

This young man was under observation for three years. At the age of twenty-five he contracted a cold which developed into acute bronchitis; on the third day he died during a fit of convulsions. No autopsy.

Considering the intensity of the lesion with the resulting pronounced symptoms, the patient's lease on life was unusually long.

Case 2.—B. F., male, 20 years old. His family history negative to communicable diseases. There were "two blue babies in the family among near relatives."

Personal History.—He was born naturally. One month after his birth, a bluish tint of his whole body was noticed. As time went on, the skin became more livid, permanent and pronounced. From his birth to the present day, his health is poor; he is tall and muscular but easily fatigued. He was always subject to cold and coughing and dyspnea. On examination the skin

of the whole body, particularly of the face and hands, had a livid tint. The mucous membranes and conjunctivæ were purplish and the eyes bulging. The fingers and toes blue and club-shaped.

The lungs showed signs of a mild bronchitis.

On percussion, the right heart was found hypertrophied. Auscultation disclosed good apex beat, accentuated aortic sound. The pulmonic sound was muffled by a loud, soft blowing murmur; the point of maximum intensity directly over pulmonic area. The murmur was carried above and below that area, but with diminished force. After severe exertion, the murmur increased in intensity, and then it could be heard between the scapulæ. There was a perceptible thrill, systolic in time, over the pulmonic area.

Liver, spleen and kidneys normal.

Examination of the blood showed 8,930,000 red cells and 160 per cent. hemoglobin.

This young man is still among the living, feels better in the summer than in the winter, and "expects to live to a good old age."

Case 3.—R. B., male, 19 years old, weight 130, height 5 ft. 7 in. Family history, negative. One aunt had a "blue" baby which died immediately after birth.

Personal History.—His birth was instrumental. Nothing abnormal was noticed about him except that, during fits of crying, his face and body got blue. He has gone through the diseases of childhood safely and was considered a strong and healthy boy. Lately he began to suffer frequent attacks of palpitation of the heart and slight dyspnea on exertion. On account of these two symptoms he came for advice. He was found of fair size and development. His skin was faintly blue; to see it, inspection had to be made in clear daylight. The mucous membranes and conjunctivæ were visibly livid. There was slight bulging of the eyes. The superficial veins of the chest were clearly defined and imparted a moderate blueness to the surrounding skin. The precordia was prominent—a condition which I observed in all the cases. Fingers and toes club-shaped. The lungs and viscera were normal. The first and second sounds of the heart were distinct. The pulmonic sound was almost hidden by a very loud systolic murmur heard best at the second left intercostal space. The right heart extended beyond the

rigth border of the sternum. A systolic thrill was felt over the pulmonic area, but not to the extent as in the two previous instances. This boy was under observation for quite a while. His palpitation improved a great deal, but the evidence of pulmonic stenosis remained.

Case 4.—A boy of eleven years. Both of his parents were rheumatic; aside from this the family history is of no importance. The little lad was never in the best of health; his main trouble was shortness of breath. His birth was natural. In his early infancy, a physician told his mother "that he has heart disease." No cyanosis of body or mucous membranes was ever noticed even during exertion.

When he came under observation he was found to be of good normal size, but of very delicate health. No bluish tint to be seen anywhere; on the contrary, the color was pale and waxy. The veins of the front of the chest were prominent. The eyes were projecting as in a mild Basedow's. The lungs and other internal organs were normal. The heart showed considerable hypertrophy to the right of the sternum. The precordia was very prominent. The apex beat and the aortic second were accentuated. The pulmonic sound was heard indistinctly. Over the pulmonic area, a violently loud systolic murmur was heard. The murmur could be heard over the whole cardiac area, but the point of greatest and loudest intensity was over the pulmonary space. The systolic thrill was palpably evident. The toes and fingers were club-shaped.

Case 5.—A girl baby about two years old. The family history is negative. The mother is rheumatic. The child's personal history is briefly as follows: birth natural; it has not been sick since its birth. It showed slight cyanosis of the body and mucous membranes since its birth. The tint is most marked when the baby cries. The eyes bulge somewhat, and the fingers and toes are club-shaped. The precordia is prominent and the border of the right heart extends beyond the right sternum. Auscultation reveals a loud systolic murmur over the pulmonary artery; the murmer is heard with diminished intensity over the cardiac region and upward to the left clavicle. There is a systolic thrill over the pulmonic area. The sounds of the heart, with the exception of the pulmonary, which is muffled, are clear and distinct.

There was no lesion to be found in any organ.

The pulse was carefully studied in all the cases, with no particular result. The pulse in pulmonary stenosis may be small or not, slow or rapid. It is influenced more by the health of the patient and bodily exertion or rest at the time of the examination, than by the lesion itself.

Having mentioned above the deciding signs which prompted the diagnosis of congenital pulmonary stenosis in our cases, it would be useless to repeat them.

The pulmonary area is, indeed, "a region of romance," or a ground for guessing; but the physical signs of stenosis of the pulmonary artery, particularly of congenital origin, are so pronounced and so unlike any other resulting from a lesion of the heart or lungs, that no mistake should be made. The only question that could arise is whether the lesion is pulmonary stenosis only or complicated by one or more forms of malformation. This question could be settled by ascertaining the point of maximum intensity of the murmur and presence of the systolic thrill. In stenosis, the two should be located over the pulmonary area. Even if suspicion be entertained of the presence of other congenital lesions of the heart besides the one under discussion, the observer is justified in disregarding them, and in view of the physical determining signs, he can say that the clinical diagnosis is pulmonary stenosis.